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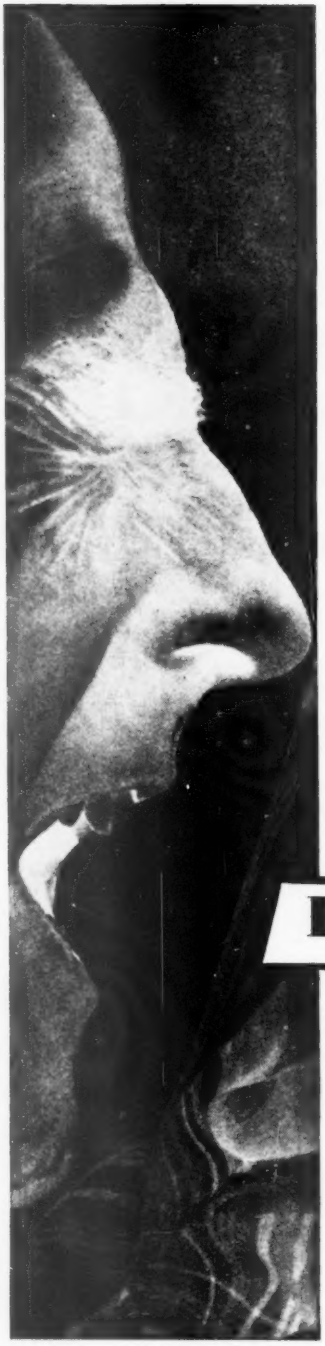

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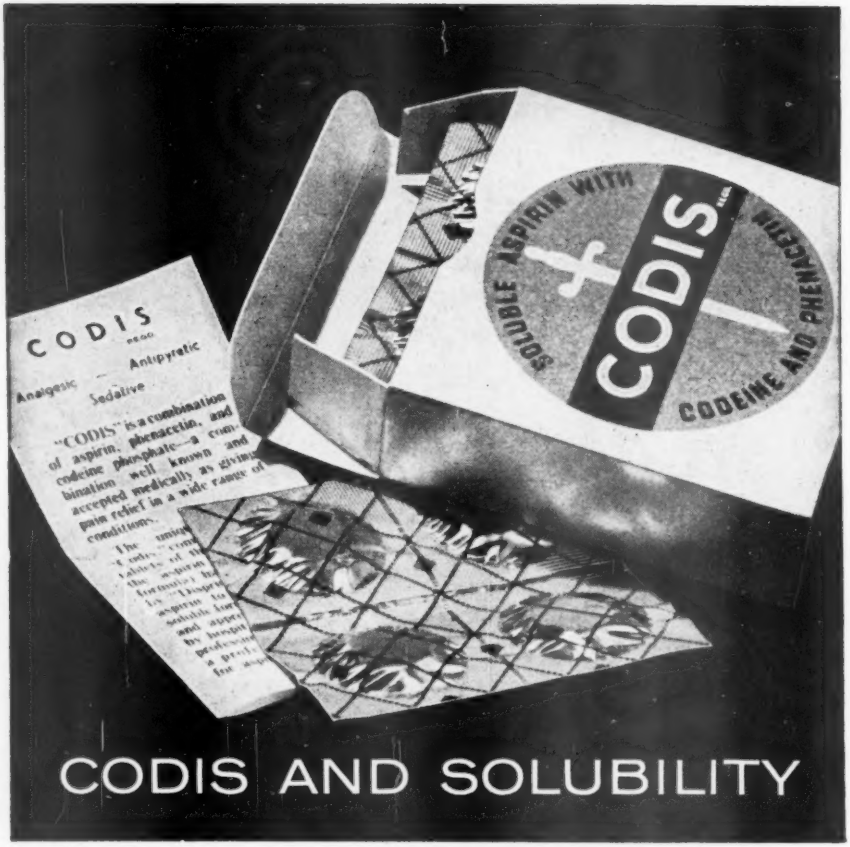

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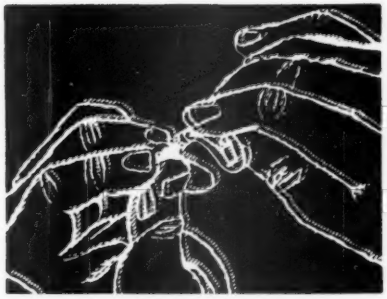

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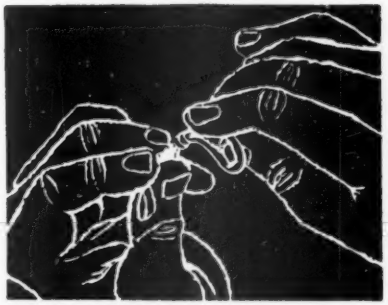

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Meeting
February 10, 1959

DISCUSSION ON INDICATOR DILUTION CURVES IN HEART DISEASE

Dr. J. P. Shillingford (London):

The Study of the Circulation by Dye Dilution Curves

Stewart in 1894 first showed that it was possible to estimate the cardiac output by injecting an indicator substance into the venous circulation, and sampling it from the arterial circulation. Hamilton *et al.* (1932) demonstrated its application in man and further showed that an estimation of the volume of blood through which the indicator had passed could be obtained. More recently the method has been used to define the position and size of intracardiac shunts (Swan *et al.*, 1957) and to estimate valvular incompetence (Korner and Shillingford, 1955). I shall indicate some of the basic principles underlying the technique and its application to the study of heart disease, while the following speakers will discuss its use in the determination of the position and size of intracardiac shunts and the practical aspects of calculating cardiac output, central volume and valvular incompetence.

Consider fluid passing from left to right through a volume represented by a glass container. If an indicator substance such as Indian ink is injected at the entrance, the particles will be spread out in their passage through the fluid. This spread may be recorded as a time-concentration curve at the exit. The parameters of this curve, in the absence of a shunt or valvular incompetence, are, to a large extent, determined by the speed of flow of the fluid and the volume through which it passes. If the speed of flow is increased the particles of indicator will reach the sampling site faster and the appearance time will be reduced; they will also pass the sampling site more quickly with a decrease in the spread of the curve. An increase in volume causes the particles to traverse more fluid and lengthens the appearance time; their dispersion is greater with an increase in the spread of the curve. It is on this principle that the study of the circulation by indicator dilution curves is based, but before going on to this in more detail I shall discuss some of the methods used for recording the curves.

Many indicators have been used by various workers in the study of the circulation in man. An ideal indicator should meet the following requirements: (1) It should be harmless when injected intravenously and cause no discomfort. (2) The amount injected should be small in bulk. (3) The indicator must remain in the blood stream and not be lost in the tissues or changed in chemical composition during its passage through the body. (4) It must be easily sampled and measured by direct or indirect methods.

Table I shows some of the methods used to record indicator dilution curves. It must be emphasized that basically, whatever the indicator and recording method used, the resultant indicator curve should be the same. At the present time both the dye and radioactive techniques are in popular use. Evans blue has become established as a satisfactory dye for obtaining dye dilution curves. The loss of this dye from the circulation is negligible over the time needed to calibrate the curve, it appears to be harmless, and its concentration is easily measured in the plasma. Its absorption spectrum (640 millimicrons), however, is in the region of that of reduced hæmoglobin; it is not, therefore, satisfactory in those patients with cyanotic congenital heart disease and a changing blood oxygen saturation. Moreover, it is only slowly excreted and stains the skin when given in large amounts. To overcome this, Fox and Wood (1957) have introduced a green dye with a spectrum absorption in the region of the wavelength 800 millimicrons, at which the absorption by oxy- and reduced hæmoglobin are the same. At present it is under trial, but preliminary reports are promising.

TABLE I

Indicator	*Method of recording*
Dyes (Evans blue, Fox green, indigo carmine, Coomassie blue)	Direct arterial puncture. Intermittent sampling. Cuvette. Photo-cell ear-piece
Radioactive tracers	Intermittent arterial sampling. Counter over heart or artery
Saline	Electrical resistance in arterial blood
Cold saline	Thermistor

More recently Coomassie blue (Imperial

Chemical Industries) (Taylor and Shillingford, 1959; Thorp and Taylor, 1959), has been found to offer several distinct advantages in that it is not toxic up to doses of several hundred milligrams, does not stain the tissue and is excreted comparatively but not too rapidly. The effects of changing oxygen saturation of the blood can be minimized by reducing the sensitivity of the recording instrument and giving larger doses of the dye.

Radio-isotopes such as radioactive iodinated human serum albumin, inorganic radioactive iodide and radioactive sodium, have all been used as tracers to produce indicator dilution curves. Unfortunately, the amount of the substance needed to produce time-concentration curves on the radio-activity recorder placed over a peripheral vessel tends to exceed the safety limit and precludes multiple injections. Estimations of cardiac output may be made by placing the counter over the heart and allowing for the passage of the indicator through both the right and left ventricles. Many early workers used the intermittent sampling technique in which blood was allowed to flow through an indwelling arterial needle into a series of tubes which collected the blood at timed intervals of one or two seconds. The development of the photo-electric cell and suitable electronic amplifiers has made possible the continuous recording of the passage of a dye. The blood may be allowed to flow continuously through a cuvette attached to a needle in a peripheral artery. On one side of the cuvette is a light source and suitable optical filter; on the other is a photo-electric cell connected via a suitable amplifier to a recorder. The passage of the dye in front of the photo-electric cell causes a decrease in the current passed and deflection of the recording pen. Wood and Geraci (1949) in America, and Kopelman and Lee (1951) and Korner and Shillingford (1955) in this country have used the ear oximeter to record dye curves in man. A light source is placed on one side of the pinna of the ear and two small selenium cells on the other side. A red filter is in front of one and an infra-red filter in front of the other. The cells are connected in electrical opposition. Changes in thickness of the ear produced by arterial pulsation affect each cell equally and are neutralized. The passage of dye through the ear affects only the cell with the red filter and produces an electrical current which may be amplified and recorded. Provided the response of the apparatus is linear, the dye dilution curve may be calibrated by measuring the height of its tail and comparing this with the final concentration in the plasma estimated spectrophotometrically.

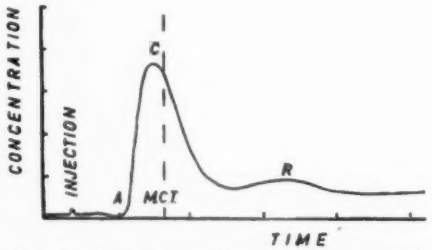


FIG. 1.—The time components of an average dye dilution curve. A, appearance time; C, time of maximum concentration; R, recirculation. (Time scale: 1 division equals ten seconds.)

Fig. 1 shows a typical indicator dilution curve obtained in a normal subject. The following measurements are those most commonly used in the interpretation of the curves: (1) The appearance time or time between the point of injection and first appearance of the indicator at the sampling site (A). (2) The area under the curve or the sum of the concentrations at unit intervals of time. (3) The mean circulation time which is the sum of the appearance time and the mean time of the curve (M.C.T.). (4) The dispersion of the particles reflected in the "spread" of the curve. This spread may be recorded as the statistical variance of the curve, as the angle of the downslope or more simply as an arbitrary fixed fraction of the maximum concentration at the peak of the curve C. (5) Abnormalities in the shape of the curve produced by the presence of intracardiac shunts. Fig. 2 shows the basic

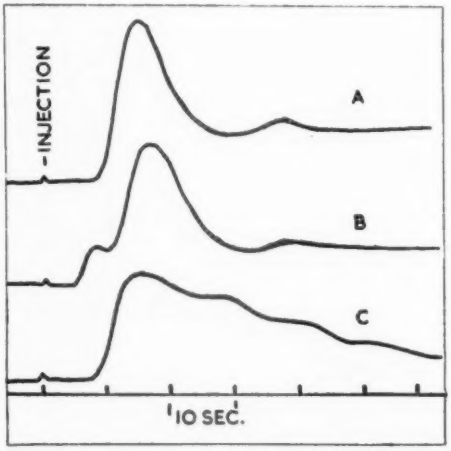


FIG. 2.—Dye dilution curves obtained from B, a patient with a right-to-left shunt; C, a patient with a left-to-right shunt, compared with one from a normal control, A.

pattern of the dye curve from a patient with a right to left intracardiac shunt. Some of the dye passes from the right to the left side of the heart ahead of the main bulk traversing the pulmonary circuit. As a result the appearance time is earlier than normal and a small primary curve appears on the upstroke of the main curve. Where there is a left-to-right shunt, part of the dye is shunted through the pulmonary vessels at each successive passage through the heart and produces a series of small curves on the downstroke of the main curve. The net effect of this is to depress the maximum height of the curve and to prolong the downstroke without significantly altering the appearance time.

Where the curve is recorded by means of a continuous photo-electric cell or radioactive counter method the concentration of dye at any point of the curve may be calibrated by estimating the concentration in a venous sample of blood taken at a point in the "tail" when all the dye has been thoroughly mixed in the blood and the curve has completely flattened out. This calibration assumes that the recording system is linear in its response.

REFERENCES

Fox, I. J., and Wood, E. H. (1957) *Proc. Mayo Clin.*, **32**, 541.

Hamilton, W. F., Moore, J. W., Kinsman, J. M., and Spurling, R. G. (1932) *Amer. J. Physiol.*, **99**, 534.

Kopelman, H., and Lee, G. de J. (1951) *Clin. Sci.*, **10**, 383.

Korner, P. I., and Shillingford, J. P. (1955) *Clin. Sci.*, **14**, 553.

Stewart, G. N. (1894) *J. Physiol.*, **15**, 31.

Swan, H. J. C., Burchell, H. B., and Wood, E. H. (1957) *Proc. Mayo Clin.*, **32**, 485.

Taylor, S. H., and Shillingford, J. P. (1959) *Brit. Heart J.* **21**, 497.

Thorp, J. M., and Taylor, S. H. (1959) *Brit. Heart J.* **21**, 492.

Wood, E. H., and Geraci, J. E. (1949) *J. Lab. clin. Med.*, **34**, 387.

Dr. Lawson McDonald (London):

Indicator-Dilution Curves in the Diagnosis of Congenital Heart Disease

New methods of investigation have particularly enriched the bedside diagnosis of congenital heart disease in the last ten years, so that an accurate qualitative and quantitative diagnosis can be made clinically in many patients. Cardiac catheterization of the right and left sides of the heart may still be necessary to confirm the clinical diagnosis. Indicator-dilution techniques are now a useful adjunct to cardiac catheterization; they often make angiocardiography unnecessary, and offer a method in research which can yield new information. Wood and Swan at the Mayo Clinic have led the development of dilution techniques in the diagnosis of congenital heart disease, and much of what I shall say stems from their work, which has recently been summarized (Symposium, 1958). The apparatus used at the Institute of Cardiology consists of a light source with a sealed infra-red and red-sensitive barrier-layer assembly (Norman, 1959). It may be used either as an earpiece, or as a cuvette, and a flow of air at 4° C. maintains the complete unit in thermal equilibrium. The output from the photocells is directly conducted to a Honeywell-Brown recorder, via a simple balancing network. The frequency response of the whole apparatus is limited only by the recorder. The dyes which we use as indicators are T. 1824 (Evans blue), indigo carmine, Coomassie blue and cardiogreen. Either Coomassie blue or cardiogreen is preferred for cyanosed patients.

Practical aspects.—The ear must be warm and flushed if it is used as the sampling site. Radiant heat or a suitable ointment should be applied to it for sufficient time to produce maximum vasodilatation. Adequate time must also be allowed for the instrument to warm. Uniform dilution curves with a steady base-line will not be obtained unless these precautions are observed. Sampling with an earpiece is usually adequate, although a cuvette may sometimes register minor changes in the curve more certainly. But sampling with a cuvette may be essential, as in selective sampling from various chambers of the heart. The position of the catheter should be checked by fluoroscopy, both before and after each injection or sampling; it may slip from one site to another, especially in the hyperdynamic heart.

The use of this method may be considered under four headings: (1) Qualitative diagnosis of left-to-right and right-to-left shunts. (2) Quantitative estimate of the size of shunts; this is described by Shillingford and Cliffe. (3) Identification of vessels and chambers. (4) Research purposes: for instance we have recently made some simple observations by this method on the velocity of pulmonary blood flow in atrial septal defect (McDonald *et al.*, 1958, 1959).

Qualitative diagnosis of left-to-right shunts.—If dye injected on the right side of the heart participates in a shunt of blood from left to right, the resulting dilution curve is qualitatively altered. This occurs in patent ductus arteriosus, and atrial and ventricular septal defects. Dye injected into the right ventricle in atrial septal defect participates in the left-to-right shunt and the dilution curve is characteristically altered, whereas dye injected into the left ventricle does not, and the curve is normal in shape (Figs. 1 and 2). The dilution curve of left-to-right shunt shows a maximum concentration of dye

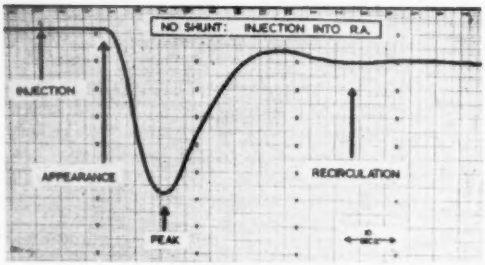


FIG. 1.—Normal dye-dilution curve after injection of dye into the right atrium and sampling at the right ear. In Figs. 2 to 10 sampling is also at the right ear.

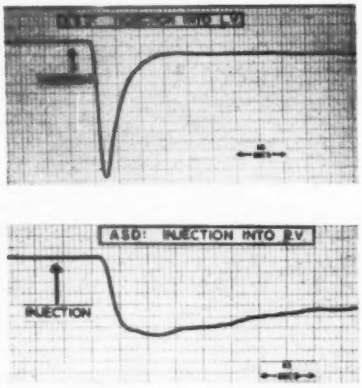
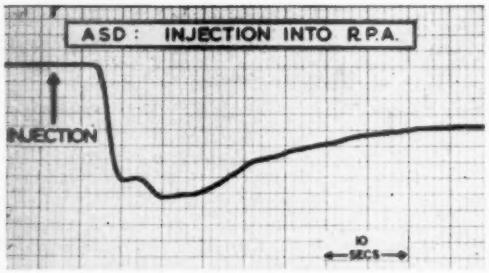


FIG. 2.—Atrial septal defect (ostium secundum) with left-to-right shunt. Dye injected into the left ventricle (above) shows a normal dilution curve, and into the right (below) the changes of left-to-right shunt.



←FIG. 3.—Atrial septal defect (ostium secundum) with left-to-right shunt. After injection into the right branch of the pulmonary artery, the dilution-curve shows a double hump and the pattern of left-to-right shunt (*see* text).

which is less than normal, a more gradual downslope, and a disappearance time that is disproportionately increased. These changes are due to the slow clearance of dye from the central circulation: during each passage of dye through the left heart some escapes to the right, and so there is in effect a series of small curves.

In most cases of atrial septal defect more blood from the right lung than from the left takes part in the left-to-right shunt. Thus a curve of left-to-right shunt is usual after injection into the right branch of the pulmonary artery, whereas injection into the left may produce a relatively normal curve. In addition the dilution-curve after injection into the right branch of the pulmonary artery not infrequently shows a double peak (Fig. 3). This is due to some blood draining from the site of injection into the right atrium, and the remainder entering normally into the left atrium.

If dye is injected into a branch of the pulmonary artery, beyond the level of a left-to-right shunt, and blood sampled from a systemic artery and from different sites in the right heart, the level of the shunt may be shown. Thus in ventricular septal defect, if blood is sampled from the right ventricle, dye appears on the dilution curve earlier than on a curve from a systemic artery, whereas the appearance time from the right atrium is not shortened in this way.

The dilution curve returns to normal after abolition of a left-to-right shunt, as by the surgical closure of atrial septal defect (Fig. 4).

Qualitative diagnosis of right-to-left shunts.—If dye is injected into the right side of the heart at the level of, or proximal to, the site of a right-to-left shunt, it joins in the shunt, and partly escapes to the left side of the heart. The resulting dilution curve shows a relatively short appearance time and a double hump (Fig. 5). The short appearance time is due to blood directly entering the circulation of the left heart and appearing early at the sampling site. The double hump is caused by the two different initial passages of dye through the circulatory system; in one dye passes through the normal route and in the other it escapes to the left side of the heart. The dilution curves are different when dye is injected at, or proximal to, the level of the right-to-left shunt, from those obtained after injection distal to this site (Fig. 6). When dye participates in the right-to-left shunt the curves declare it, whereas the distal curves are normal in shape. Thus the site of the defect is shown.

Identification of vessels and chambers.—A vessel or chamber may be identified by comparing the characteristics of a dilution curve

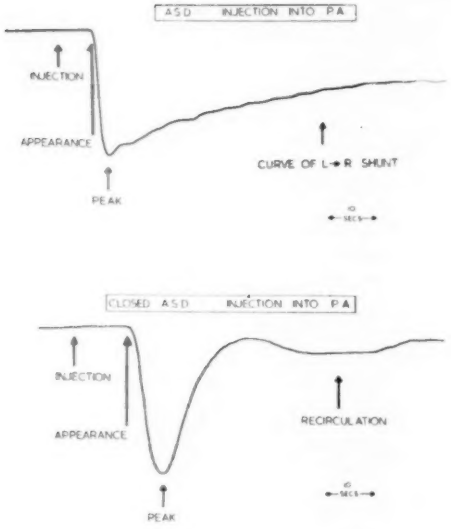


FIG. 4.—Atrial septal defect (ostium secundum) before and after surgical closure. Dilution curves after injection into the pulmonary artery show abolition of the pattern of left-to-right shunt after surgical closure of the defect. Reproduced from McDonald *et al.* (1959) by kind permission of the publishers of the *British Heart Journal*.

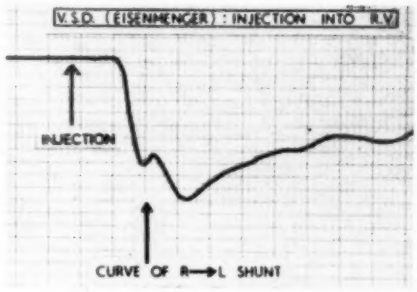


FIG. 5.—Ventricular septal defect (Eisenmenger syndrome). Dilution curve shows the double hump of right-to-left shunt.

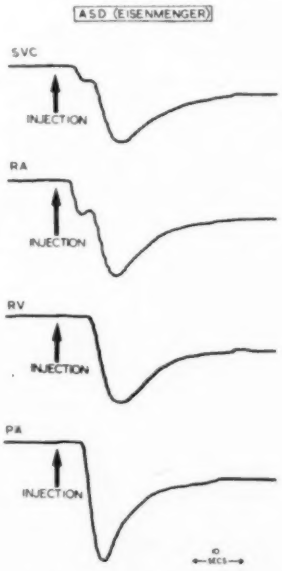


FIG. 6.—Atrial septal defect (Eisenmenger syndrome). Dilution curves after injection into the superior vena cava (SVC) and right atrium (RA) declare the right-to-left shunt. Curves after injection into the right ventricle (RV) and pulmonary artery (PA), i.e. beyond the level of the shunt, are normal.

obtained from it with others from known sites of injection. Dilution curves obtained from injection of dye into an atrium, or vessels adjacent and draining into it, are similar to each other; they differ from dilution curves obtained from comparable sites on the opposite side of the heart. Thus curves following the injection of dye into the right atrium, and vessels that drain into it, in atrial septal defect, contrast with those from the left; they are qualitatively different, and the appearance time is longer than on the left side (Fig. 7). A cardiac catheter may enter the left atrium, and obviously pass thence into a normal pulmonary vein of the left lung. But the catheter may enter the middle lobe of the right lung in a position compatible with a pulmonary vein that either enters normally into the left atrium, or anomalously into the right. If dye is injected into the vein, and a dilution curve recorded, it may be shown to be either normal or anomalous. The dilution curve after injection into an anomalous pulmonary vein is similar to those obtained after injections into the superior and inferior venæ cavæ (Fig. 8). On the other hand, dye injected into a normally draining pulmonary vein results in a curve which is different in shape from those obtained after injections into the venæ cavæ, and its appearance time is shorter (Figs. 9 and 10). Vessels and chambers may similarly be identified in more complex abnormalities, such as the various forms of transposition of the great vessels.

Conclusion.—These examples show how indicator-dilution methods enable certain vital

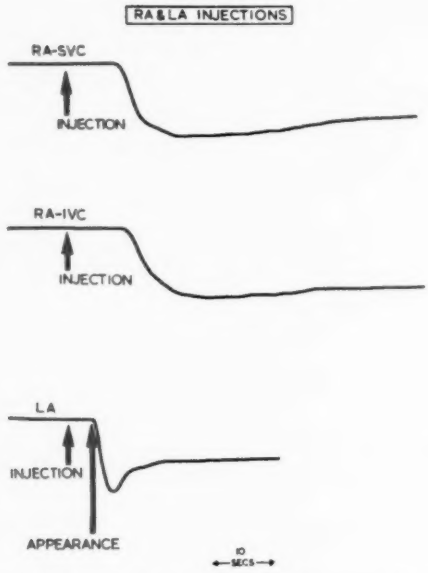


FIG. 7.—Atrial septal defect (ostium secundum). Injections of dye into the right and left atria, and their adjacent vessels, yield curves which are qualitatively different, and the appearance time is shorter on the left side.

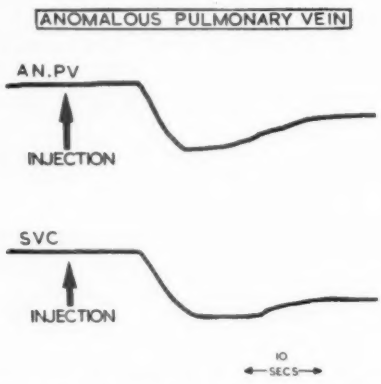


FIG. 8.—Atrial septal defect (sinus venosus type). Dilution curves after injection into an anomalous pulmonary vein (AN.PV) and the superior vena cava (SVC) are similar in shape.

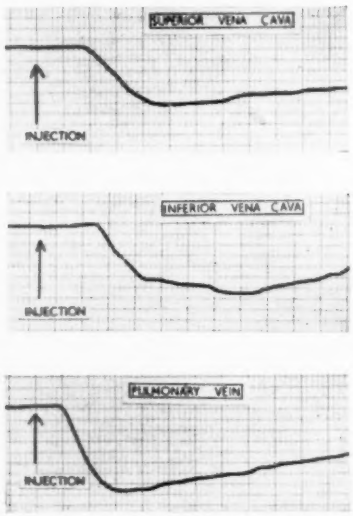


FIG. 9.—Atrial septal defect (ostium secundum). Dilution curve after injection into a normally draining pulmonary vein is different from curves obtained after injections into the superior and inferior venæ cavæ.

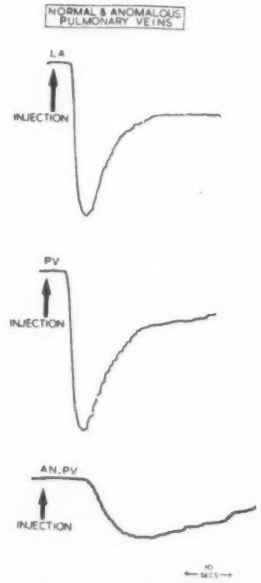


FIG. 10.—Atrial septal defect (sinus venosus type). Dilution curves after injections into left atrium (LA) and a normally draining pulmonary vein (PV) are similar, and contrast with a curve after injection into an anomalous pulmonary vein (AN.PV).

information, in the diagnosis of congenital heart disease, to be obtained both simply and safely.

REFERENCES

McDONALD, L., EMANUEL, R., and TOWERS, M. (1958) *Brit. Heart J.*, **20**, 586.
——, ——, —— (1959) *Brit. Heart J.*, **21**, 279.
NORMAN, J. (1959) *Brit. Heart J.*, **21**, 107.
Symposium on Diagnostic Applications of Indicator-Dilution Curves Recorded from the Right and Left Sides of the Heart (1958) *Proc. Mayo Clin.*, **33**, 535.

Dr. P. Cliffe (London):

Quantitative Aspects of Indicator Dilution Curves

Three quantities may be calculated from a normal indicator dilution curve: (1) Cardiac output. (2) The mean circulation time. (3) The central volume.

Calculation of cardiac output.—Hamilton *et al.* (1928, 1929, 1932) have shown how to calculate cardiac output. Consider a normal dye dilution curve, Fig. 1A. The first concentration peak is due to dye which has passed the sampling site on the first circulation, while the second peak is due to recirculation. The effect of recirculation must be eliminated to calculate cardiac output. The smooth downslope of the curve is exponential in form, i.e. if plotted as logarithm of concentration against time, the downslope becomes a straight line. The effect of recirculation is eliminated by assuming that the exponential downslope would continue unchanged if recirculation did not occur. The dotted portion of Fig. 1A represents the remainder of the primary curve. In Fig. 1B the dye curve is replotted on semi-log paper and the exponential downslope becomes a straight line. This line is continued to the base-line and while the recirculation curve is present it is separated from the primary passage of dye. The base of the curve now becomes the time taken for dye on the first circulation to pass the sampling site. A simple numerical example (Hamilton, 1953) makes the method clear. Suppose that the dose of dye injected is 12 mg., and that the average concentration of dye (average height of dye curve) is 4 mg./litre. The dye must therefore have been diluted by 3 litres of blood; this occurs in the thirty seconds required to sweep out the curve, i.e. the cardiac output is 6 litres/minute. Generalizing this example:

$$\text{Output} = \frac{60.12}{4.30}$$

$$= \frac{60.\ \text{Dose}}{\text{Average Concentration} \times \text{Time}}$$

$$= \frac{60.\ \text{Dose}}{\text{Area under Curve}}$$

$$\text{Further Average Concentration} = \frac{\text{Sum of Concentrations}}{\text{Number of Concentrations}}$$

where the sum is made at equal intervals of time. If the interval is one second, the number of concentrations is equal to the number of seconds, i.e.

$$\text{Average Concentration} = \frac{\text{Sum of Concentrations at 1-second intervals}}{\text{Time}}$$

Thus,

$$\text{Output} = \frac{60.\ \text{Dose}}{\text{Area}}$$

$$= \frac{60.\ \text{Dose}}{\text{Sum of Concentrations at 1-second intervals}}$$

The latter expression gives the usual practical method. The sum of concentrations at one-second intervals is calculated from the semi-log replot and divided into 60× Dose. Actually, the dye is carried by the plasma and not by the erythrocytes so that in the above expression, the plasma output is multiplied by H/(100 − H) where H = corrected venous hæmatocrit, to give cardiac output. The standard deviation between the dye method and the direct Fick method has

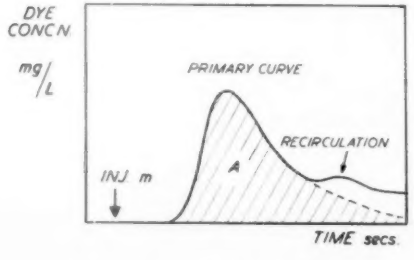


FIG. 1A.—Normal dye dilution curve.

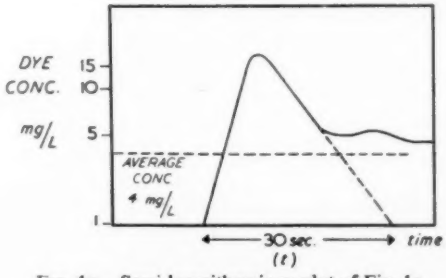


FIG. 1B.—Semi-logarithymic re-plot of Fig. 1A.

frequently been shown to be approximately 20% and no systematic difference has been demonstrated between the methods.

Calculation of mean circulation time (M.C.T.).—This is the average time for a dye particle to traverse from injection to sampling site, i.e. imagine 1,000 particles in the injection, and that the time for each could be measured from injection to sample. The sum of these times divided by 1,000 is the mean circulation time. It is calculated from the curve, since

$$\text{M.C.T.} = \frac{C_1t_1 + C_2t_2 + \ldots\ldots}{C_1 + C_2 + \ldots\ldots\ldots}$$

i.e. the sum of the product of concentrations and the corresponding times at 1-second intervals divided by the sum of concentrations. Times are measured from the instant of injection of the dye.

Calculation of the central volume.—Central Volume = MCT × Cardiac Output.

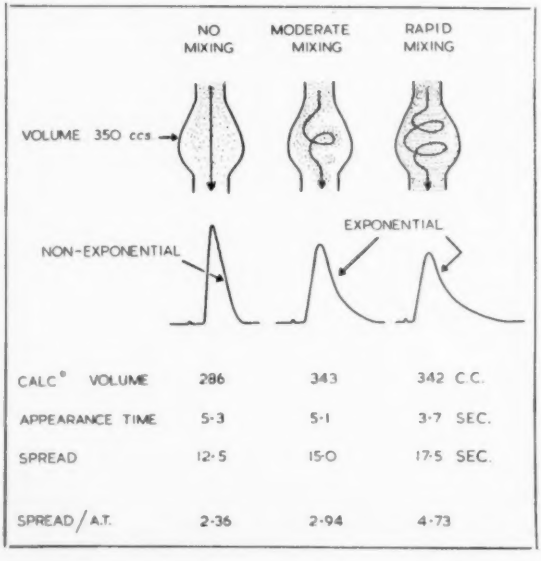
In the case of venous injection with arterial sampling, this volume is simply the volume of blood leaving the heart during the mean circulation time. Regarding the anatomical meaning of this volume, suppose the injection is via the antecubital vein. Dyed blood passes centrally and when it reaches the right heart it is met by venous blood from all those venous pathways throughout the body equidistant in time from the right heart. The dyed blood passes out into the arterial system and reaches the sampling site. Simultaneously, dyed blood reaches all other points in the arterial tree equidistant in time from the centre. The central volume is the sum of all these venous and arterial volumes and is thus diffusely distributed throughout the body.

The exponential downslope.—Emphasis has been placed on the significance of the exponential downslope of the dye dilution curve. If it be absent, the form of the primary circulation cannot be predicted and the calculation for cardiac output fails. It is natural to enquire as to what type of system would lead to the exponential downslope. A model comprising a central mixing chamber with water flowing through it will do so when dye is injected, especially if the mixing process is complete in the chamber. Hamilton and his co-workers (Kinsman, 1929) showed that a heart-lung preparation yielded an exponential downslope when used as the model, but it is unlikely that more than a moderate dispersion of dye occurs in such a preparation or in man.

Fig. 2 illustrates a definitive experiment due to Shillingford. It shows a chamber of volume 350 c.c. in which the rate of mixing can be varied. Water flows through the system at a constant rate and the pathway of injected dye can be followed by electronic flash photography. Results show:

(*a*) *With no mixing:* (1) The downslope is not exponential. (2) The calculated central volume (Hamilton) is much below the actual volume.

(*b*) *With moderate mixing:* (1) The downslope *is* exponential. (2) The calculated volume closely



←FIG. 2.—Effect of mixing on dye curves.

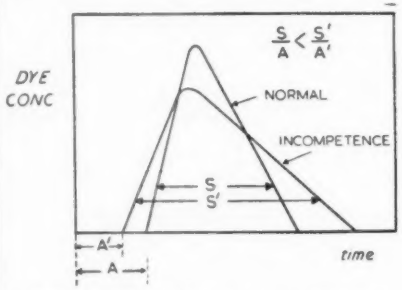


FIG. 3.—Effect of valvular incompetence.

approaches the actual volume. (3) The spread of the curve is increased. (4) The appearance time is decreased.

(*c*) *With complete mixing:* The features in condition (*b*) are accentuated.

Summarizing, the exponential downslope of the dye curve allows the cardiac output to be calculated and suggests that sufficient dispersion of the dye is present to validate the calculation of volume. It is probable that in man, dye curves which lack such a downslope will lead to considerable errors in computation.

Application to valvular incompetence.—The mixing experiment of Fig. 2 has elucidated the effect of valvular incompetence on the form of the dye curve. The regurgitant jet of blood acts like the stirrer in the model and disperses dye particles forward and back. While the Hamilton calculations are still valid, the shape of the curve has changed in that the spread has increased and the appearance time has decreased. This is shown in Fig. 3. A convenient way of measuring the spread of the curve (Shillingford, 1958) is as the width of the replotted log curve in seconds at a height of one-tenth the maximum concentration. In absence of incompetence, spread is *increased* by either a decrease in cardiac output or an increase in central volume. However, these effects are accompanied by an *increase* in the appearance time. The ratio Spread/A.T. thus remains sensibly constant. On introducing valvular incompetence the spread increases but the A.T. decreases. Thus the ratio Spread/A.T. increases. Shillingford (1958) has shown that this ratio correlates closely with clinical assessments of the severity of mitral incompetence.

REFERENCES

HAMILTON, W. F. (1953) *Circulation*, **8**, 527.
——, MOORE, J. W., KINSMAN, J. M., and SPURLING, R. G. (1928) *Amer. J. Physiol.*, **84**, 338.
——, ——, ——, —— (1929) *Amer. J. Physiol.*, **89**, 331.
——, ——, ——, —— (1932) *Amer. J. Physiol.*, **99**, 534.
KINSMAN, J. M., MOORE, J. W., and HAMILTON, W. F. (1929) *Amer. J. Physiol.*, **89**, 322.
SHILLINGFORD, J. (1958) *Brit. Heart J.*, **20**, 229.

Meeting
March 10, 1959

The Professorial Medical Unit

PRESIDENT'S ADDRESS

By M. L. ROSENHEIM, C.B.E., F.R.C.P.

THE Haldane Commission on University Education in London, in its report issued in 1913, recommended the establishment of whole-time units in several schools. In the evidence given before this Commission by Abraham Flexner, and quoted in the Goodenough Report, appear the following criticisms of medical education in 1913:

> "Clinical teaching in London remains an incident in the life of a busy consultant" and
> "No certain reward stimulates the young physician to engage in original work. His cue is faithful routine. In consequence his preliminary scientific training goes largely to waste."

Following the publication of the Haldane Report, the Faculty of Medicine in the University of London, at a meeting held in 1914, expressed the opinion that

> "... The University atmosphere would be best created in a Medical School by the encouragement of those who seem to possess the necessary qualifications to pursue research untrammelled by the necessity for earning their daily bread, and that this end could be obtained with the least disturbance of existing methods by the appointment in each Medical School of one or more professors of advanced medical subjects. These professors should devote considerable time to the organization of research, and should give a certain amount of teaching of the academic type. The more technical aspects of the student's education would be left in the hands of the present clinicians, who should be encouraged and aided in their endeavours to advance the knowledge of the subject which they teach".

University College Hospital and Medical School accepted proposals put forward by the University for the establishment of medical, surgical and obstetric units, each directed by a whole-time professor, but the war intervened and it was not until 1920 that clinical professorial units were started in several of the London medical schools.

Nearly forty years have passed since these units were founded with the objects of the integration of teaching, the encouragement of research and an attempt to bring the teaching of

medicine into line with that of other scientific disciplines in the University.

At the time of the Goodenough Report, in 1944, only five out of the twelve medical schools in London had full-time professorial undergraduate chairs in medicine and there were only three such chairs in the provincial schools of England and Wales. To-day eight of the London schools have such chairs, one has a chair of experimental medicine, while one of the remaining three has established a medical unit to which, in due course, a professorial chair is likely to be attached. In the provinces there are now eight whole-time undergraduate chairs of medicine, and during the same period whole-time chairs have been established in many of the medical schools throughout the Commonwealth.

This is an impressive change, and whole-time units are now generally recognized as playing an important role in undergraduate and postgraduate medical education, though their function and responsibilities are not always appreciated and are sometimes misunderstood.

I should like to take this opportunity of paying tribute to Professor T. R. Elliott who, as the first whole-time professor of medicine at University College Hospital, directed the medical unit there for some twenty years. While Sir Thomas Lewis was widening the scope of clinical research, and demonstrating the possibility of scientific experiment on man, T. R. Elliott was shaping the future of academic medicine in this country. The ever-increasing number of professorial units, and their record of teaching and research, bear eloquent testimony to his vision, enthusiasm and guidance.

There are, I believe, four main duties of such a unit:

(1) Care of patients.
(2) Research.
(3) Undergraduate teaching.
(4) Training of postgraduate research workers.

Care of Patients

There are striking differences between the professor of medicine in Great Britain and those on the Continent or in the United States. Here the professor is not in charge of the whole medical clinic, but is a physician to the hospital in line with his part-time colleagues, though he usually has more beds under his care. I am, myself, in favour of this system.

The number of beds allotted to the unit should be sufficient to provide the professor and his established staff with adequate clinical experience and research facilities. If the assistants on the unit are to be given gradually increasing clinical responsibility, if students are to be given sufficiently wide experience of medicine and if a number of beds are to be set aside for research into certain specific diseases, forty to fifty beds should prove sufficient. Such a firm will require two registrars and two house physicians who are normally provided by the hospital.

The medical unit must always aim to set a very high standard of medical care. I am a firm believer in the importance of the continuity of medical care and cannot reconcile myself to the American system under which the immediate care of the patient is in the hands of the resident, with a rotating team of interns, while the senior staff go on "full duty" with charge of a ward for two to three months at a time. It is true that such a system does permit more doctors to have the privilege of charge of beds and frees time for research, and that an exchange of duties among assistants on the unit is necessary from time to time, but I doubt whether frequent changes are to the advantage of the patient, the ward or the junior staff.

The assistant on the medical unit must receive gradually increasing personal responsibility for the care of patients. This will, at first, be in the outpatient department, later he may be given a few beds for his special research cases and ultimately he should have general beds of his own.

It is vitally important, on a large unit with many assistants and research assistants, that it is always made quite clear who is in charge of any individual patient, and that that doctor should direct treatment and discuss diagnosis and progress with the patient. The patient will be seen by many other people, including the professor, but the line of contact must be clear and must be respected, or else the patient will suffer. The patient undoubtedly benefits from free discussion of diagnosis and treatment by members of the staff, but he must know who is his doctor and he must be seen regularly by him. We do not always pay sufficient attention to ensuring that the patient knows who is who in the hierarchy of the ward.

The care of the patient must always come first. The unit must acquire a high reputation for the practice of medicine, and I would, of course, include here the social care of the patient. Unless this is so, not only will teaching suffer, but the unit will be regarded as a place where research is more important than the patient.

Interest in special diseases necessitates the establishment of clinics where the progress of patients can be observed. Such follow-up clinics have often been started as part of a research project and have proved extremely valuable. One of the main duties of an academic unit is the application of new knowledge to the

practice of medicine. This may involve the investigation of new laboratory procedures or the trial of new methods of treatment. Once a new test has been proved of value, it must be taken over by the hospital laboratory, while a special clinic which becomes firmly established should also pass from the control of the unit to that of the hospital. Otherwise the man-power of the unit soon becomes absorbed in routine duties.

The ward sister in a professorial unit is a most important person and has a vital, but difficult, role to play. While she must try to help the various members of the unit in their special projects, in balance studies, special investigations and in the trial of new drugs, and must be accurate and scientific in her observations, she must always see that the patient is not over-taxed, over-examined or questioned, and that he or she gets sufficient rest. The ward sister is an essential member of the team and must be kept informed of all that goes on.

Teaching

In England the professorial unit would never claim to hold the prerogative of teaching. Traditionally a great deal of teaching, both in the wards and lecture room, is done by our part-time colleagues, many of whom take endless trouble over their work. An increasing number are, of course, specialists, with highly expert knowledge of certain aspects of medicine and complex methods of investigation and treatment and many devote a major part of their time to their hospital work. The place of the specialties in undergraduate teaching is still unsettled. In so far as the student sees sick patients fully investigated, and sees research in progress, he cannot fail to benefit from clerking to a special firm, but he should not be exposed to the teaching of detail that belongs to special postgraduate study. This is a difficult problem, and the allocation of students for their clerking must be carefully controlled.

The professor may organize the set teaching in the school, special demonstrations and conferences, but bedside teaching remains all-important and cannot be directed or supervised. The professor should try to integrate the curriculum, should be able to introduce experimental teaching methods and should ensure the teaching of the scientific background of medicine. To this end, I believe that the organization of the introductory clinical course is one of his most important duties.

The whole-time staff have the best opportunity of contact with members of the preclinical departments and such contacts must be encouraged, with the exchange of ideas, combined research projects and, of course, co-operation in teaching. We are fortunate, at University College Hospital, in having a senior lecturer in applied pharmacology and therapeutics who is attached both to the Department of Pharmacology and to the Medical Unit, while we also have close and effective contact with the biochemists and with the department of genetics.

Most professors during the past year have been involved in that ancient, but somewhat overrated, pastime of trying to revise the curriculum. The new regulations of the General Medical Council give wide scope for experiment in medical education and I hope that universities and schools are really going to experiment. A major difficulty in education is the impossibility of devising a controlled trial. Something will, however, be gained if medical education ceases to be a "double blind" procedure, and if both teachers and students observe and report on the results of such experiments. While the curriculum has changed very little, the actual education that the medical student of 1959 receives has altered markedly from that of 1929, mainly in the realization that both the patient and the student are human beings under stress and that personal relationship plays a large part in the treatment of both.

The main contribution that the whole-time unit makes to medical education is by research. We are, or should be, training students in scientific methods and criticism and so preparing them for the advances that are to come. They must understand how such advances are brought about, how clinical problems can be scientifically investigated and new drugs accurately tested, and they must appreciate how relatively little of our present knowledge rests on sure scientific foundation. It is by association with people engaged in research, by being present at clinical experiments and by seeing new methods of treatment under trial, that this attitude of mind is formed. The participation of students as subjects in clinical experiment is of great educational value, but I would stress the term participation, which implies a clear explanation and discussion, once the experiment is over, of its objects and results. The ethical aspects of clinical experiment and trial, both on patients and students, must always be freely discussed, and must always remain the ultimate responsibility of the professor.

The Staff

The staff of a unit consists of the established university assistants, of research assistants from this country or overseas, non-medical graduates and technicians.

Each unit normally has a fixed establishment of

university assistants, and their welfare, education, promotion and future form one of the main professorial duties. It is now generally agreed that a unit requires a second-in-command, a deputy director, reader or senior lecturer, who is of consultant status, and who has full charge of beds and can assume direction of the unit. The unit of the future may well need both a professor as director, and a research professor, as well as a number of senior lecturers. While the reader has a lifelong appointment and receives full staff status, the first assistant or senior lecturer, usually a five-year appointment, receives an honorary contract as a consultant in the hospital, but is not regarded as of full staff status. Even if not given such status in the hospital, senior lecturers should play a part in the school organization and should be active members of departmental committees.

Selection of Staff

At a time when opportunities for advancement in clinical research or in academic medicine are limited, the appointment of an assistant to an established university post is a major step. Such appointments now usually go to people who have demonstrated their ability to do research, and who have often been qualified for five years or more. Even though the appointment is officially renewable (and therefore terminable) annually, it is a mistake to appoint an untried man to such a post. Granted that the new assistant has already shown his ability, and he must of course already be a Member of the Royal College of Physicians, there is much to be said for making the appointment for an initial period of two or even three years. This allows the assistant time to settle down and to undertake some long-term investigation, while it places a barrier ahead when his future must come up for serious review. Annual reappointment tends to be casual and automatic, and the assistant may become relatively senior before his capabilities and future are discussed. A longer original appointment provides him with a period of security and the knowledge that reappointment for a further term will only occur if he has made good.

If only more senior men are appointed to established posts, provision must be made for training the younger. Every academic unit should be able to carry 3–4 junior research assistants, and funds should be available either from internal research funds or from outside sources to enable the professor to test out young men with ideas. Such research assistants may come straight from pre-registration posts, or may have completed some years as registrar.

Money is no longer the limiting factor to recruitment for research. The Medical Research Council has scholarships for training in research, and also makes grants to established workers to enable them to have assistance. The problem is one of selection.

It is as difficult to select the good research worker from among recently qualified doctors as it is to select good students from the applicants for admission to medical schools. In both cases, the good man usually selects himself. The future clinical scientist has often already had some research experience in the basic sciences, or has become interested in some problem and presents himself with a request for opportunity. In every batch of clinical students there are some who have spent an extra year on a preclinical subject and who have some research experience, and such students should be encouraged, whenever possible, to maintain their interest in research during the clinical course.

I believe that every registrar should be encouraged to have some research project. This may be the testing of a new drug or the elucidation of some physical sign, or he may work as a member of a team tackling some more complex problem. The physician of the future should have insight into the difficulties, as well as the satisfaction, of research. The academic units can do much to foster research among registrars, and from their ranks may come recruits for further training.

The junior research worker may have a skill ready to apply, but more often he will need apprenticeship and guidance. He may have some specific problem in which he is interested and may be anxious to "go it alone", but it is often better for him to start by working with a more experienced person, learning methods and techniques. He must remain clinically orientated, for he may well find that he has taken the wrong turning and may wish to return to a clinical post. Even though his work lies in the laboratory, he must be encouraged to join discussion rounds in the wards and at least once a fortnight should accompany the professor or his deputy on a full ward round. Attendance at grand rounds should be considered as important as the monthly meetings to discuss research projects.

After one or two years the young research assistant may be ready to take advantage of the Medical Research Council clinical fellowships in research, and may go to spend a year or more working under an expert in a preclinical or clinical department before he returns to the unit as an established assistant.

Here I should like to digress for a moment, to point out that while units differ in their size and form, they fall into two main groups as regards

research activity: the unidirectional and the diffuse. In some units almost all research is directed at one major problem, while in others each assistant has his own special interest, though these often overlap and allow team work. I believe that the latter affords more scope for training young men, and it certainly makes for more interesting and lively discussion in the wards.

To return to the established assistant, appointed after a period of trial, already equipped for research, with a problem he wants to tackle; while he must be given increasing clinical responsibility, he must have adequate time for research, and must be provided with adequate space, with the equipment he needs and with technical assistance. If he is to do effective research, teach and accept some clinical responsibility, he must have either the full or part-time help of a technician. Later, a graduate biochemist or biophysicist may be needed to complete his team. He must himself be experienced in all the chemical or electronic methods that he uses, but he should be able to pass work of a routine nature to a reliable assistant.

So far, so good; but what of the future of these established assistants? They must, in due course, move on or all recruitment and promotion will cease. Some will become consultants in either teaching or non-teaching hospitals, but many wish to remain in academic medicine, in clinical research and teaching.

In his memorandum to the Goodenough Committee, Sir Thomas Lewis, discussing the recruitment of workers and the organization of research, stated that:

> "When a worker has proved his research ability, has chosen research as a career, and has been accepted for such, he should be given reasonable security in this career. This means that he must be able to see before him the prospect of increasing facilities and responsibilities, provided by appointments, until he reaches a level where his abilities find full scope and where his remuneration is adequate."

One of the major problems at the present time is the limited number of posts to which the trained research worker in medicine can graduate. These include full-time academic appointments (and these are slowly increasing in number) and research posts in Medical Research Council or other special units. There are, unfortunately, too few posts in which the responsibility of the care of patients is combined with the opportunity and facilities for continued research.

The majority of doctors who, at the age of 26–28, embark upon research, are not aiming at a full-time research career. They are interested in clinical medicine, in the work of the physician, and would ultimately like to hold an independent post with clinical responsibility and the opportunity of research. It was this type of post that many of us hoped to see established by the National Health Service, and which we still hope may be encouraged and supported by the Clinical Research Board. It is the existence of such posts, in abundance, in the United States that is one of the main reasons for the vitality and rapid advance of American medicine. Every university hospital in America has a body of young physicians who devote the major part of their day to research.

Medicine is advancing more rapidly than ever before, new fields are opening up, new methods of investigation and treatment appear daily, but such advances and their application depend upon men trained in research and equipped with special techniques. In this country we are training an increasing number of first-class men. We are encouraging them to devote themselves to the investigation of vitally important, but intricate, problems, but we are not providing sufficient senior posts to which they can graduate.

Many are unlikely to continue indefinitely in research, but when they are first appointed to the consultant staff of a hospital, be it a teaching or non-teaching hospital, they should be given time and facilities for research. Otherwise their expensive training is wasted. There is a great need for such posts—part-time clinical and part research, and this need is not appreciated by our Boards of Governors or Regional Boards. In their absence, we are not making proper use of the men we train.

Professor Peart (1958) has published some striking figures, as a result of writing round to various academic units. He obtained the following figures from 15 medical units in England, Scotland and Wales for a period of ten years:

Assistants from Medical Units passed on to:

Consultant posts	52
Chairs, Readerships or Senior Lectureships	37
Emigrated	14
Research posts	10
	113

From the Postgraduate Medical School:

To teaching hospitals or research posts	18
To regional consultant posts	13
Emigrated	13
	44

It is not easy to analyse these figures in detail. Professor Peart points out that in ten years, 50% have moved on to consultant posts, 25% to more senior academic posts and 17% have emigrated. If we take the 113 who have advanced from the 15 undergraduate medical units, this gives a

turn-over of 7·5 men per unit in ten years, or 3 men leaving each unit every four years, which is a surprisingly high figure. Some 50%, however, of the trained workers go to posts in which they are unlikely to continue to do research. It should be possible to provide such men with time, laboratory facilities and salary to allow them to continue, at least for five years, work which can only be to the benefit of the hospital to which they have been appointed.

We cannot analyse further the fate of those who have emigrated, but some have gone to academic posts in the Commonwealth or in the United States, to posts for which they are admirably suited by their training.

We have, of course, a duty towards the Commonwealth and this brings me to the last major point which I want to discuss—the relationship of the academic unit in this country to schools overseas. I am sure that we would all agree that professorial units should, within the limits of their facilities, welcome research workers from overseas, postgraduates who come to join in research and who often contribute greatly to the work in progress. Such visitors, from the Commonwealth, from Europe or from the United States are usually financially supported by their own University, by the British Council or other fellowship and, provided that they settle down to work, they present no problem. We welcome them and we learn from them. We would also, I am sure, agree that young doctors who come to this country only to study for higher degrees or to obtain clinical experience, should not be attached to academic units. There is a third category of visitor, the senior teacher, the professor or reader from a Commonwealth university, often in one of the less developed countries, who can gain a great deal from short-term attachment to a unit. Those of us who have seen the very difficult and different conditions under which they have to work would always welcome them and give them the opportunity of seeing how our schools are run, how the teaching is organized and what research is going on. Such a visit may prove a great stimulus not only to the visitor, but to his school on his return.

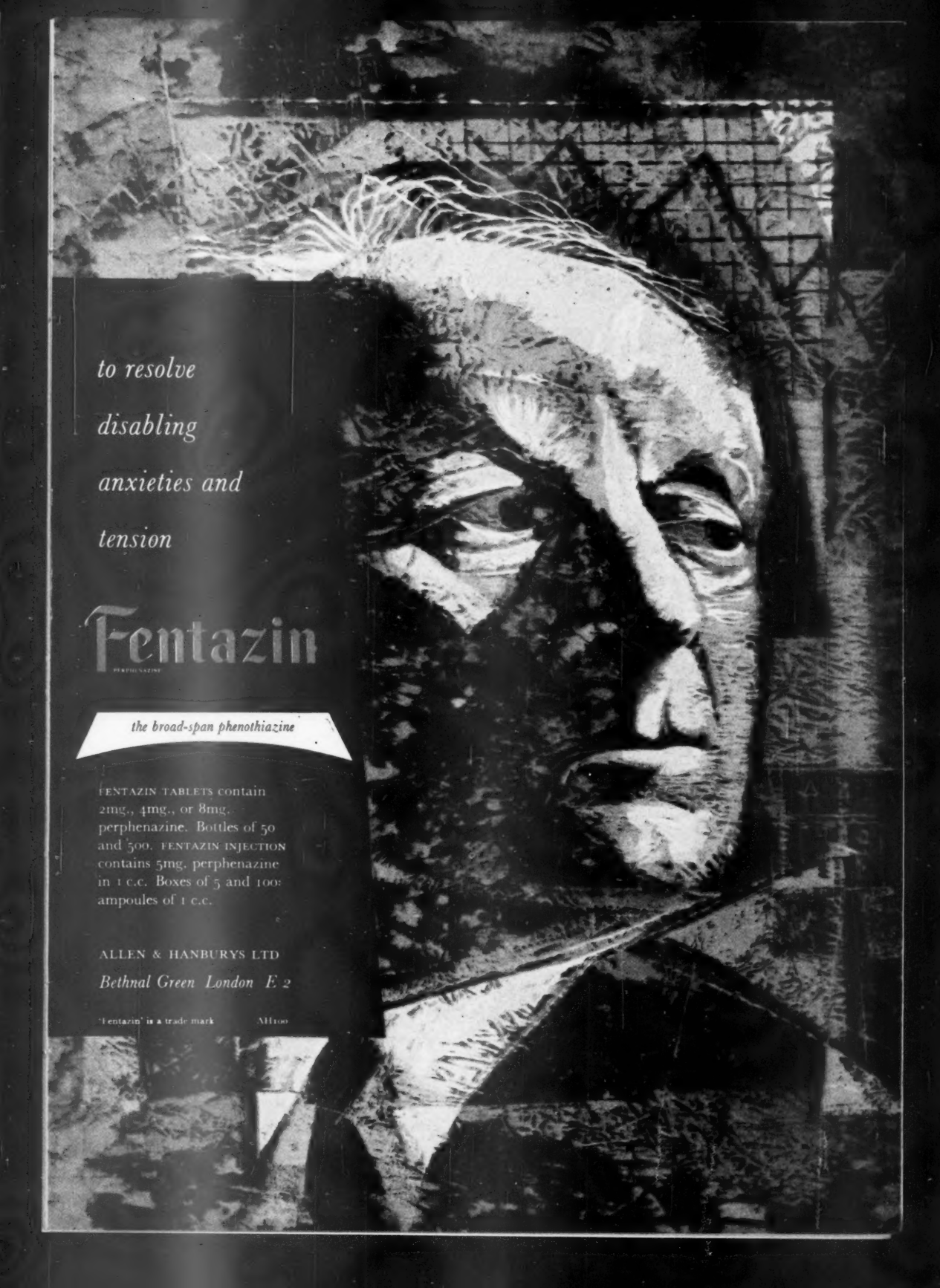
I need hardly say anything about the value of travel to the assistants on a unit, for this is now well recognized, and a year of work in a first-class department in the United States or elsewhere is an invaluable part of their training. Many of us wish that we could organize some means of exchange or secondment with the newer medical schools of the Commonwealth, but there are still many difficulties. I hope that such exchanges may become possible in the future.

There are many other problems that face the academic medical unit and the Professor of Medicine. I should like most warmly to recommend Fuller Albright's classical Presidential Address which he gave in 1944 to the American Society for Clinical Investigation. It is entitled "Some of the 'Do's' and 'Do-not's' in Clinical Investigation". In the course of his Address, Albright says: "See to it that you do not wake up some fine morning in an executive job." In other words, don't become a professor of medicine. It is difficult for the professor not to become involved, at least to some extent, in committees and administration, and while it is a fascinating and rewarding job, most professors of medicine with whom I have discussed this matter feel frustrated by the multiplicity of their duties and by their inability to do all of them effectively.

What of the future? This country requires experts in medical research as well as engineers, chemists and atomic physicists. The present rate of medical advance calls for more and more men trained in scientific thought and methods, and it is to be hoped that the Universities, the Teaching Hospitals, the University Grants Committee and the Medical Research Council will realize that we cannot afford to fall behind in medical progress or to waste the fully trained men produced in our academic units.

REFERENCES

ALBRIGHT, F. (1944) *J. clin. Invest.*, **23**, 921.
PEART, W. S. (1958) *Lancet*, ii, 1232.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[1] Rosa, L., *et al.*, *Acta allerg.* (Kbh.), 1957, **XI**, 81

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Section of Laryngology

President—R. L. FLETT, F.R.C.S.

Meeting
March 6, 1959

DISCUSSION ON RESPIRATORY CATARRH IN CHILDREN

Professor Douglas Hubble (Birmingham):

Ætiological Factors

All children catch colds—an incidence of three or four a year is the average endowment in childhood—but, fortunately, in only a minority of these children is an acute infective or allergic episode associated with them. Fewer children still develop a chronic catarrhal state but there are enough of them to crowd our out-patient clinics and to make a not inconsiderable addition to the sum of human anxiety and misery. The catarrhal state is a complex of constitutional and environmental factors and he is a bold and uncritical man who would maintain that he can apportion accurately the blame for these disorders.

It may not be altogether inappropriate to liken a chronic catarrh to one of our turgid, sluggish and muddy Midland rivers oozing its way through lush and overgrown meadows to a final estuary escape. How much have the tributaries of infection and allergy contributed to this pestilent stream? How much responsibility has the nature of the river-bed itself? What has been added to the effluent by the filth of our city atmosphere?

Although it would be wrong to pretend that we can provide exact answers to these questions, it is necessary for us to make an attempt to assess ætiological factors in the management of the individual child.

The infective factor.—This we must regard as the first cause and, even where allergic, environmental and local factors have acquired importance, infection and its control must be our primary consideration. These other causes are important because they condition an increased liability to infection. Consider the environmental situations within the family which encourage infection. Overcrowding increases the risk of infection and the return of some members of the family from their community life outside the home—buses, trains, schools, factory, offices—creates an additional infective hazard for the young. The psychologists speak, and rightly, of the vulnerability of a child's situation in the family order, and in infection the physical vulnerability of the younger children is not only *relatively* increased by the return of the workers but may also be *absolutely* increased by the increased virulence of an organism in its passage through several hosts.

Probably most of the acute infections of the nasopharynx, larynx and trachea are of viral origin and our attack on these invaders is limited by the lack of anti-viral agents. We are better equipped to deal with bacteria, and since any extension to ears and to bronchi may be due to these secondary invaders we should treat these children in whom otitis or bronchitis is the recurring sequel to a nasopharyngeal infection with antibiotics—promptly and efficiently. The intelligent mother (and maternal wisdom is now more highly rated than when I was a young doctor) should, under the supervision of the family doctor, be given either phenoxymethylpenicillin or tetracycline tablets to keep in the house to administer to her susceptible child at the first onset of a cold. If this *prompt therapy* is not effective, and sometimes it is not, in preventing the acute complication, then it is worth while to give continued *prophylaxis* by penicillin tablets either G, 200,000 units twice daily, or V, 60 mg. twice daily. This is essentially a prophylactic method of preventing the *complications* of acute infections.

It by no means excludes another sensible approach to the problem of the child with the recurring acute catarrhal infection, which is to allow the episode to proceed without intervention by antibacterial therapy, in the hope that the child's immune processes will thereby be encouraged rather than recurringly inhibited, while reserving the antibacterial agents for the treatment of complications. This method has not been scientifically assessed; but it is rational and it frequently succeeds, in individual cases, in reducing the frequency of acute infections. Not until these three alternative methods of prompt treatment, prophylaxis and the judicious neglect of therapy, have been thoroughly tried (unless, for social reasons, they are inapplicable) should nose and throat surgery be undertaken for the prevention of the recurrent acute infection.

The chronic infective catarrhal state with mucopurulent nasal discharge or with cough associated with infected sputum requires the consideration of allergic and environmental factors, but it also requires from the physician a definition of the nature and extent of the damage

to the respiratory tract, sinuses, ears, bronchi and lungs. Here he will require the help of the otolaryngologist, the radiologist and the bacteriologist. The respiratory tract is functionally indivisible and to talk of upper respiratory catarrh as distinct from lower respiratory catarrh is nearly impossible; the pædiatrician is fortunate in that his territory, unlike that of the oto-rhino-laryngologist, is not given an anatomical definition. Consider the difficulties which are imposed on your specialty by the definition of the larynx as your distal boundary. There is the problem, for example, of whether the sinuses are secondarily infected in the bronchiectatic child (as most of us believe), or whether (and I know that some of you hold this opinion) bronchiectasis is frequently the sequel to sinusitis. Bacteria know no boundaries and we should then encourage both the thoracic surgeon who betrays an interest in the sinuses, and the laryngologist who dares to use the bronchoscope; just as we should applaud both those general and thoracic surgeons who regard the diaphragm as a hurdle and not as a hedge. These surgical buccaneers may serve both our science and our patients.

Bronchiectasis is, of course, a common accompaniment of infective catarrh, and this diagnosis justifies the use of antibiotic prophylaxis, in addition to the usual physical measures. Three rare conditions which the pædiatrician will not overlook, when he is considering the chronic catarrhal child, are hypothyroidism, of which upper respiratory catarrh may be an obvious manifestation; agammaglobulinæmia in which there is a defective resistance to recurring acute infections; and mucoviscidosis. Mucoviscidosis, in hospital practice, is by no means an uncommon finding in the catarrhal child. The thick tenacious secretions which are found in the trachea and bronchi produce emphysema and atelectasis as the first changes. The child has a chronic cough; repeated acute infections supervene with bronchopneumonia and bronchiectasis. There is a congenital abnormality of the mucous glands in all situations; and, in addition to the pancreas, the mucus-secreting glands of the nose, the sinuses, the trachea and the bronchi are also affected. It is not uncommon now for the skilled pædiatric radiologist to give a report on a chest X-ray in some such terms as these, "these lung changes are consistent with fibrocystic disease of the pancreas".

To consider treatment in mucoviscidosis, these infections should be prevented by continued tetracycline therapy, and we are now using neomycin inhalations in older children. The prevention of infection gives these children a much better prognosis, for their survival after infancy depends on the extent of the naso-pharyngeal, bronchial and pulmonary lesions.

The diagnosis is made in hospital by demonstrating a deficiency of pancreatic enzymes in the duodenal juice, using trypsin as the indicator of the reduction of all pancreatic enzymes. Even the sweat glands are affected in this disease and this has introduced another useful screening test in the diagnosis of mucoviscidosis, for the analysis of the sweat shows an increased concentration of chloride, sodium and potassium.

The allergic factor.—The rhinologist may feel confident when he looks inside the nose and sees a pale hypertrophied mucous membrane that he is seeing the result of an allergic process, but if he then hands the child to the pædiatrician with a request to identify and remove the offending allergen, he is giving him a tough assignment. Most of us are content to attempt desensitization with stock dust extracts in non-seasonal allergic rhinitis, and to use pollen extracts to prevent summer exacerbations. Beyond this we use the general prophylactic and therapeutic measures which we employ in asthma.

This we recognize to be a superficial approach which is damned by the allergists. However, I read in preparation for this paper a recent essay by an American pædiatric allergist on allergic problems in the upper respiratory tract and I feel bound to say that it provided very little assistance, either in the diagnosis or management of these catarrhal states when approached from the allergic standpoint. We move down the respiratory tract only to encounter another diagnostic and therapeutic problem in allergy which by no means relieves our feelings of physicianly inadequacy. It is the problem of the child who develops a "wheezy" chest every time he catches a cold. Is he suffering from asthmatic bronchitis or spasmodic bronchial catarrh? Has this wheeze any relation to allergy?

To answer these questions we depend on a family history of allergic manifestations (though as Frankland pointed out the role of heredity in allergy has never been satisfactorily established), on a history from the patient which appears to incriminate an allergen, and on the presence or absence of eosinophilia. The fact that there is some response to antihistamine therapy does not establish an allergic pathogenesis; for the antihistamines will by a non-specific effect (? an atropine-like action) on both the nasal and the bronchial mucosæ reduce swelling and turgescence. Whether or not an allergic process is involved makes no difference, we suspect, to the pathological changes in the bronchial mucosa.

If the antihistamines give a useful therapeutic result then it is reasonable to advise the family doctor to use not only an antibacterial agent but also a sympathicomimetic drug, like ephedrine,

or an antihistamine, in conjunction with the antibiotic at the first onset of the catarrhal process.

Disappointment is often expressed at the results to be obtained from such drugs whether in prophylaxis or in treatment; this is sometimes justified since the effective therapeutic result is accompanied by disturbing side-effects. More often the lack of therapeutic success depends on the failure to recognize that the correct dose of these drugs demands individual assessment. Here there are no fixed rules for prescribers such as we may provide for antibacterial agents. Some children require, and can tolerate, astonishingly large doses; and these remedies should not be discarded until they have been used in doses large enough to produce side-effects. There is now a great range of antihistamines, all of them effective but in varying dosage, and the prescriber is well advised to employ only one or two of them so that he becomes thoroughly familiar with their usage.

Environmental factors.—I use the word "environment" here to include both the internal and external *milieux*, all the factors which we assume to be contributory to the child's catarrhal state and which facilitate the infective and allergic factors already discussed.

It is customary for the textbooks to state that attention must be paid to the child's hygiene, though precisely what can be done in this regard and, if improved hygiene is achieved, what benefit is to be expected, is not usually stated. Most of these catarrhal children are adequately nourished, and if they are not, it usually depends on a failure of appetite consequent on the catarrhal state. Many of them live in the great cities in overcrowded conditions, and despite the slum clearance and the new building there has not, owing to the steady increase in population, been much improvement in overcrowding in the last twenty-five years. The increased exposure of the younger children in the family to infection is not easily preventible and although it might be thought that we increase this hazard by expelling them from their homes, at an early age, to nursery schools, yet for those whose homes are overcrowded and whose mothers go out to work the nursery school may indeed diminish the liability to infection. Smokeless zones in our cities will never give us an atmosphere as clear as that of the country or the seaside, and I believe firmly that it is valuable to send these catarrhal children, even for short periods, to the sea or the country.

I have not found that psychological factors are of importance in the ætiology of the chronic catarrhal state as they sometimes may be in the acute recurrent allergic and infective catarrhal disorders.

Effects of chronic catarrh.—Here it is appropriate to discuss the results of chronic catarrh in the child, for it is in a consideration of the ætiological environmental factors that we see that cause and effect become inextricably entangled. The catarrhal state becomes established in the early years at school but after the age of 8–9 many children happily escape from it, we assume because of their improved resistance to infections. In the unfortunates who remain the victims of chronic catarrh, cough, lack of appetite and insomnia supervene — with pallor, inadequate growth and failure to make average weight gains as the insignia of the catarrhal state.

It can be well understood that depression is engendered in the child and anxiety in the parents and that these emotional reactions may have their influence on the child's physical condition. Depression and anxiety do not in this case require psychotherapy—apart from the encouragement that is derived from the knowledge that interested experts are doing their best to help. For ourselves there must be the determination to interrupt the cycle by physical treatment, and I believe that this can as a rule be accomplished providing that local disease is not too firmly entrenched.

Local factors.—I shall leave my colleagues to deal with the management of the local situation in the catarrhal state, stating only my opinion that surgery should not be undertaken until the surgeon is satisfied that the family doctor or the pædiatrician has done all that may be medically accomplished to prevent the recurring acute or allergic episode and to diminish the miseries of the chronic catarrhal state.

These miseries can hardly be overestimated; to live each day in the wretchedness that we have all intermittently experienced in the acute cold surely justifies the continuing enquiry which, as I know, some of the members of this Section are giving to these disorders. Although there have been these advances in medical therapy, both prophylactic and curative, I have been able to say little about the ætiology and pathogenesis of catarrh in childhood that might not have been said twenty-five years ago and therefore the studies which Mr. A. Tumarkin and Mr. H. Zalin have been pursuing in Liverpool have a peculiar importance both for oto-rhino-laryngologists and for pædiatricians.

The medical contribution of the last twenty-five years lies in the wise use of the antibacterial agents, the sympathicomimetic drugs, and the antihistamines—in themselves no mean achievements. A great step forward will occur when we achieve effective anti-viral prophylaxis and therapy.

Mr. H. Zalin (Liverpool):

Several years ago the Liverpool Regional Hospital Board, being alarmed at the excessive waiting list for children's tonsillectomy, decided that a special unit should be created to deal with the problem, and in due course an obsolescent fever hospital, later named the John Bagot Hospital, was allocated for that purpose. For several years I had already been doing similar work for the Liverpool Education Authority and had been very dissatisfied by its restricted nature. My appointment to the John Bagot promised facilities which previously had been lacking and stimulated my interest in the whole problem of upper respiratory disease. No one, I think, will deny that the so-called tonsil and adenoid problem is but one facet of a vastly complex syndrome, and the more I studied it the more complex it seemed to become. Certainly so far as ætiology is concerned it is necessary to think not only in terms of the whole respiratory tract, both upper and lower, but also of more general factors which at first sight seem very remote from our own specialized territory.

For example Lawther (1958) relates bronchitic illness more closely to atmospheric pollution than to any other factor. Ogilvie and Newell (1957) describe chronic bronchitis as a social disease with a strikingly greater impact on unskilled as compared with professional workers. Oswald (1958) shows the highest mortality rates from respiratory infection to be in the industrial regions of Yorkshire and Lancashire, the rural districts of the south and west being much less affected. Gorham (1958) shows a close correlation between bronchitis and the acidity of urban precipitation. He attributes the low pH value of the atmosphere in industrial areas to hydrochloric acid and states that aerosol droplets may at times exhibit pH values less than 2.

Thus, in general, it appears that lower respiratory disease is closely correlated with industrial poverty and I submit that a similar correlation exists between upper respiratory disease and industrial poverty. Tumarkin (1957) has shown quite clearly that children of the artisan class exhibit far more ear disease than children of the middle class. Within recent years Abercrombie (Medical Research Council Survey, 1957), Fry (1958) and others have described their experiences with various aspects of upper respiratory disease. In general they conclude that it is a benign condition. As I shall show later my own experiences are much less cheerful and the only explanation I can find for this contradiction is the fact that my study in the main deals with the slums of a great city in the north whereas these authors have spoken of more fortunate patients in the south.

Again, when I embarked on this work years ago, I thought it was in the main a problem of bacterial invasion, and of course with the advent of the antibiotics I looked forward confidently to a prompt and facile solution to our problem. I need hardly say we have been profoundly disappointed. The contrast between our success with acute pyogenic disease in general and our failure with this upper respiratory disease emphasizes the complex nature of the problem. It is not simply a matter of recurring bacterial invasion. We have to consider the soil and the seed.

What do we know of the nasal bacteria? It is agreed that whilst the upper reaches of the nose are sterile the respiratory regions usually carry a varied bacterial flora. Fabricant (1941), however, maintains that where the pH is on the acid side, i.e. about 5·5 to 6·5, the whole nose is sterile. This is an important statement to which I shall return. Masters *et al.* (1958) showed that pneumococci and hæmophilus are important in nasal disease just as in chronic bronchitis. In healthy adults these organisms are relatively rare. Staphylococci and hæmolytic streptococci also have their highest incidence in school children. In addition to these more virulent organisms there are the familiar non-pathogens. In our children it is quite exceptional to find a sterile nose; moreover, not only is the flora at any given swabbing liable to be multiple but it seems to change from day to day, so that when we contemplate preparing an autogenous vaccine we take at least six swab specimens over an interval of two weeks. We used to distinguish sharply between true pathogens such as *Streptococcus hæmolyticus* and non-pathogens such as *Staph. albus*, *Micrococcus catarrhalis*, &c., but now it is generally recognized that the distinction is by no means watertight, and we ourselves believe that under the special conditions that obtain in these children even comparatively innocuous organisms can join in the attack.

So much for the seed; what of the soil? We are driven to postulate certain potentiating factors and under this heading I include (1) poverty, (2) atmospheric pollution, (3) viruses, and (4) allergy.

The effect of poverty is seen at its most tragic when large families with feckless parents are crowded together in single rooms. Such groups seem to run their own private epidemics. Thus whilst in better-class families the danger period occurs about the age of 5, i.e. when the child enters the overcrowded schoolroom, in the poorest classes the child may be at risk almost from birth. I have already outlined the evidence incriminating atmospheric pollution. How could this factor work?

There are two obvious possibilities. The pollution might act as a direct irritant. An

extreme example of this is the spontaneous perforation of the nasal septum which is said to occur in chromium workers. More interesting, however, is the possibility that the effect might be indirect—some processs which might favour the secondary invasion of bacteria by a weakening of the natural defences.

There are two main natural defences in the nose: (1) lysozyme, (2) the ciliary mechanism.

Lysozyme was studied by Fleming (1929) in 1929. It is highly bactericidal. It is found in high concentration in tears, nasal mucus, sputum and other tissue fluids. Fabricant (1945) declares emphatically that it works best in a pH of 6·5 to 7 and that it loses much of its potency in an alkaline medium of pH 8 or more. This fits well with his statement that when the nasal pH is 6·5 the nose is sterile.

You may imagine our excitement when we discovered that almost without exception the nasal pH of the slum child was 8 or more. Here, we felt, must be the mechanism of this general breakdown in the nasal defences. The nose, we assumed, in reaction to the acid irritation of the atmosphere throws out a defensive alkaline secretion; this reduces the efficiency of the lysozyme and so paves the way for secondary bacterial invasion. We thereupon set out to test the theory. We tested a large group of middle class children in Liverpool and were not surprised when they too yielded a figure of 8. After all they too are submerged in the same atmospheric pollution of our great city. We then wrote to our friends abroad. Professor van Dishoek in Leyden kindly undertook to examine a group of children in a clean rural area of Holland, Professor Kristensen kindly provided similar figures from Denmark and we ourselves examined 90 children from several schools in Bala, North Wales. A similar investigation was performed by Dr. Varnavides in Cyprus.

You may imagine our chagrin when every group yielded the same value as our own slum children, i.e. about 8. Whatever the mechanism whereby atmospheric pollution predisposes to respiratory disease it does not appear to be a simple shift in ionic reaction. This conclusion is of course tentative. Our investigations are being extended with more careful evaluation of various factors such as climate, seasonal variations, technique of pH measurement, &c. Meanwhile we are apparently confronted by a sharp contradiction between our own value of 8 and the widely quoted figure of 6·5–7 given by Fabricant (1941), Tweedie (1934) and others. Probably, as Parkinson (1945) points out, the values obtained vary considerably with the technique adopted.

These findings, of course, do not invalidate our original suggestion. The evidence incriminating atmospheric pollution still remains, even though the mechanism remains obscure.

Quite apart from this atmospheric breaching of the mucosal defences there is the well-known effect of the viruses. In coryza, influenza and similar diseases there is a primary viral invasion followed by a secondary bacterial invasion which can seriously augment the duration and severity of the initial disease. We cannot help suspecting that a similar process is at work in these children and we have carried on a good deal of investigation in an effort to pin-point it.

Recent advances in virology emphasize the complexity of the problem and expose our ignorance of the basic facts. Certain viruses have, of course, been fairly clearly identified, for example, the adenoviruses. When these were first recognized we thought they must surely play some part in this syndrome since they are so frequently located in the adenoid itself. However, they characteristically produce short, sharp pyrexial episodes which are followed by a demonstrable rise in antibody titre and a subsequent immunity to further infections of that type. In our children there is little evidence of acquired immunity and certainly we have not, as yet, succeeded in demonstrating any raising of the antibody titre. Immunity, if it does develop, does so very slowly. Thus it does not appear that we can incriminate these adeno-viruses—an unfortunate conclusion since vaccines can be prepared against them. Instead we are driven to postulate an unknown virus which does not, apparently, produce any natural immunity. Can it be that the invader is none other than the elusive coryza virus which, as is well known, produces no demonstrable antibodies? Very recently hæmadsorption techniques have been used to identify para-influenzal viruses in a study of various types of respiratory disease in poor-class children below the age of 2 (Chanock *et al.*, 1958, 1959). There is strong evidence that these viruses may produce, or help to produce, a wide range of clinical syndromes.

Finally in considering possible background factors we cannot overlook allergy. A definite proportion of our cases present the characteristics of so-called allergic vasomotor rhinitis. The slum child is no less subject to nasal allergy than his more fortunate brethren. We see the effects of a combination of atmospheric insult and virus and bacterial invasion with an explosive reaction of the nasal mucosa to inhaled allergens. The result is often pitiful. The relatively small nasal fossa of the child becomes totally occluded by a pale œdematous mucosa. The lower respiratory tract may add its quota of bronchospasm to complete the picture of respiratory distress. The inferior turbinals particularly become the seat of

permanent hyperplastic change. The addition of an allergic diathesis to a problem already sufficiently intractable makes treatment notoriously difficult and disappointing.

Thus, excluding allergy and poverty we incriminate three basic factors in the pathogenesis of this syndrome: (*a*) atmospheric pollution, (*b*) virus invasion, (*c*) bacterial invasion.

Both (*a*) and (*b*) separately or together are regarded as lowering the mucosal resistance to (*c*). Alternatively (*a*) may pave the way for (*b*) which in turn opens the gates for (*c*). This, at any rate, is how I view the initial stage of the disease when the child is suffering with recurring colds and intermittent nasal obstruction. I suspect that the actual pyrexial episodes are of viral origin whilst the continuing smouldering catarrh is the result of bacterial action plus atmospheric irritation.

Later when tonsillitis, otitis and other complications develop, pyrexial episodes may well be due to primary bacterial invasion, although even so I suspect that the viruses continue to contribute to the basic syndrome.

In general, therefore, I find it helpful to think of the disease in three stages:

(1) The initial stage of recurring colds with variable catarrh and intermittent nasal obstruction.
(2) The stage of established lymphoid hyperplasia.
(3) The stage of complications: (*a*) sinusitis, (*b*) otitis, (*c*) lower respiratory disease (bronchitis, bronchiectasis).

I have already dealt with Stage 1. Stage 2, of course, is the basis of the great tonsil and adenoid problem—established lymphoid hyperplasia (defence mechanism)—toxic absorption—mechanical obstruction. Here we encounter the perennial question of when to operate. In any given case side by side with the benign defence hyperplasia there are the deleterious effects of toxic absorption and/or mechanical obstruction to be evaluated. A case which remains in Stage 1 will usually subside spontaneously when the child reaches the age of 8–10. Even Stage 2 shows a considerable tendency to spontaneous regression, a fact which is reflected in the modern conservative attitude to tonsillectomy. With Stage 3, however, the situation alters very much for the worse. The child is heading for disaster.

For example, I believe that much of the sinusitis of adult life is laid down in childhood. X-rays in these children almost invariably reveal thickened mucosa and not infrequently frank opacity. In 100 such cases under the age of 10 subjected to proof puncture by the Watson-Williams technique we found clean antra in 60%, the polyp sign in 22% and frank pus in 18%. Thus 22% of these patients show evidence of hyperplastic changes involving the sinus mucosa. This condition may well be the precursor of chronic low-grade sinusitis in adults. Can it also explain the obscure catarrhal conditions, vague headaches and nasal obstructions which puzzle us so often and are variously diagnosed as metabolic allergic, endocrine, functional, &c.? Even more important is the group with frank pus in the antra. No less than 18% were thus affected. 12 of the frankly infected cases were submitted to chest X-ray and 6 of these had bronchiectasis. None of these children had hitherto been suspected of harbouring any chest pathology.

We regard this finding as indicative of a diffuse breakdown in respiratory resistance. I now therefore, X-ray the chest whenever an antral irrigation reveals frank pus. By this routine we hope to diagnose early cases of bronchiectasis at a stage when the chest symptoms are relatively few and the condition has accordingly been unsuspected.

The pathogenesis of ear disease is not in doubt. It stems from the two main factors of eustachian obstruction and infection, and its pathognomonic stigma is hypocellularity. The evidence for these assertions has been marshalled by Mr. A. Tumarkin in his well-known debates with Dr. M. Diamant (Diamant 1940, 1949). The basic conceptions of his neo-Witmaackian theory, are that the process of pneumatization which begins at birth and normally progresses until about puberty, becomes inhibited when air entry is prevented by obstruction of the eustachian tube. In addition the smouldering infection of the nasopharynx only too readily finds its way into the devitalized middle-ear cleft. This conception of eustachian obstruction, with or without superadded infection, seems to me so plausible that I have been surprised to find some authorities doubting it. Unfortunately the clinical examination of eustachian function is not so simple as it is generally assumed to be. Information derived from the eustachian catheter can be misleading. Only too often it is impossible to elucidate the significance of the sounds one hears with the auscultation tube. Recently Mr. Tumarkin has devised a simple technique for overcoming that difficulty. A small glass tube fitted with a rubber tip is inserted into the external auditory canal. A drop of paraffin in the outer end forms a meniscus which moves readily in sympathy with the movements of the tympanic membrane. Actually this is no new discovery as the textbook by Milligan and Wingrave (1911) illustrates a similar technique and Pollitzer himself used something of this nature. Thus it is possible to recognize many striking variations in the behaviour of the eustachian tube. Some tubes are widely patent,

others behave in a valvular manner, whilst others are completely blocked. Catheterization does not necessarily reveal the action of the tube under normal conditions. A tube which is impermeable to the catheter is probably impermeable in daily life, but the converse is not necessarily true. A tube may be permeable to the catheter and yet remain closed in daily life, just as in the case of the urinary bladder. We are studying this problem with other methods based on the work of Zollner, van Dishoek and Kristensen and we hope to describe that work in detail at some later date. It will suffice here to say that we are abundantly satisfied that eustachian malfunction plays a major role in the production of oto-pathology.

We do not know the exact rate at which oxygen is absorbed from the tympanic cavity but a few figures may be of interest. Atmospheric pressure equals about 1,000 cm. of water of which oxygen contributes about 20%, i.e. 200 cm. of water. But with our tympano-dynamometer we found a definite displacement of the drum in response to a pressure difference of 2 cm. of water. Thus the absorption of 1% of the intratympanic oxygen will cause a significant shift of the drum. It is not surprising, therefore, that persistent eustachian blockage has these disastrous results. I must emphasize, however, that this is a highly complex process, partly mechanical but also, in varying degree, inflammatory. The inflammation itself varies from the fulminating violence of a streptococcal invasion to a sullen seepage possibly suggestive of a virus.

Above all, however, I would emphasize that, so far as the ear is concerned, this is frequently a silent disease. It is true that violent septic episodes are common and that many children report varying degrees of otalgia; nevertheless in a surprising proportion of cases advanced pathology is found without any history whatever of past ear trouble.

Finally, I suggest that it is misleading to think in terms of separate clinical entities such as exudative otitis media, blue drum, cholesteatosis &c. There is a continuous spectrum of oto-pathology between the acute suppuration at one extreme and the relatively silent exudate at the other. True, these entities all have their special characteristics, nevertheless there is a common origin and we must not be surprised to find any or all of them in combination in a given case.

Treatment

What can we do in Stage 1? Obviously local treatment to the nose offers no real solution and it is not surprising that our efforts in the past have been more or less futile. We ran the gamut of diastolization, cauterization and ionization without success. Stock vaccines also failed although recently we have again started a small pilot investigation with autogenous vaccines in chosen cases. We have never used potassium iodide or other internal medications. I have already described how with the advent of the antibiotics our hopes were raised and subsequently shattered. I do not, of course, imply that antibiotic nose drops are useless. They have a very definite, if limited application. They are particularly valuable in cases of purulent rhinitis where systemic invasion is minimal. Until recently our drops were made isotonic with saline but we now prefer them made up with Ringer's or Tyrode's solutions. Negus (1958) points out that whilst normal saline is quite incapable of maintaining the activity of excised ciliated epithelium, ciliary action under experimental conditions will continue almost indefinitely in Ringer's or Tyrode's solution. Above all we insist on a demonstration of the appropriate posture and dosage. This method, suitably applied, can give surprising—even though temporary—relief.

In a proportion of cases the progress of the disease may be halted. In many more this relief is only temporary, especially in those cases which appear to be passing into Stage 2. Where this is the case, or where the child is too naughty to accept home treatment, and above all when he is very young, we prefer to admit him to hospital where the nasal toilet is combined with systemic antibiotics, &c. Our aim must be to minimize the damage done by the secondary bacterial invasion, the virus as yet being beyond our control.

Stage 2 constitutes the basis of the familiar tonsil and adenoid problem. I will make three points concerning it:

(1) I need hardly say that we are ultra-conservative; even so we are driven to operate far too often, 500 to 600 tonsillectomies per annum. We are not influenced overmuch by recurring sore throats, but we are influenced by evidence of eustachian obstruction with secondary involvement of the ear. Thus, whenever we encounter an exudate in the middle ear we deal scrupulously with the adenoid.

(2) Paradoxically one of the virtues of tonsil dissection, as opposed to guillotine, is that it provides the profound anæsthesia which is so essential if the adenoid is to be dealt with adequately.

(3) If removal of tonsils and adenoids is really necessary it is immediately necessary. There can be no medical justification for a waiting list of months—let alone years. A child with irreversible obstruction in his nasopharynx is a surgical emergency and should be dealt with as such.

Stage 3: The problem of sinusitis calls for the most careful judgment. It is essential to dis-

tinguish between the very common minor catarrhal sinusitis and the comparatively rare true suppurative disease. The former is best left alone except in so far as it is involved in the general treatment of the first stage. Certainly direct surgery of the sinuses in this stage is out of the question. The prognosis, on the whole, is not bad, but we are deeply concerned by the possibility that some of these cases go on to develop chronic polypoidal sinusitis in adult life. We have no direct proof of this. We think it plausible and are attempting to verify it.

True suppurative sinusitis is a different story. I have already indicated our views as to its relation to bronchiectasis. I have, incidentally, confirmed its curious relation to keratosis obturans. We believe that in most of these cases there is a pansinusitis. Consequently whilst benefit may follow isolated attacks on the antra, in many cases the results are profoundly disappointing. Treatment must be directed to the underlying problems, just as in bronchiectasis itself.

The ear: In common with all other workers we are dealing with very many cases of exudative otitis media, about 150 cases per annum. I doubt if this is a new development. Like Senturia *et al.* (1958) and others we have carried out many and varied tests on this exudate and have given up the attempt to crystallize it into a special clinical entity. It is simply one feature of the great syndrome of enclosed tympanomastoiditis and its nature will vary in accordance with the nature of the parent syndrome. Careful and thorough antibiotic treatment can achieve resolution but most cases finally require aspiration through a myringotomy incision. In all cases we deal scrupulously with the adenoid, although it should not be assumed that this is the answer to the problem. Indeed not a few of our cases had already had adequate adenoidectomy.

Paracentesis suction in any case will fail if (1) eustachian function is not re-established, or (2) irreversible pathology is present in the mastoid. If (1) is not obtained, either the exudate reforms or, much worse, the drum proceeds to collapse. If (2) is present the condition recurs promptly.

In either case we consider mastoid exploration and are often dismayed by the advanced nature of the pathology encountered. This is the silent enclosed mastoiditis to which I have already referred.

At the opposite extreme lies the acutely infected ear.

One of the most tragic problems in our experience is that of acute otitis in nurslings. I am not referring to mastoiditis in association with gastroenteritis; that curious condition seems to have disappeared. We still, however, regularly encounter true suppurative otitis media in infants less than a year old—usually, I need hardly say, in poor-class families. Such patients must be immediately hospitalized, not only to ensure adequate treatment, but also to get the child away from the seething infection of the overcrowded tenement. The suppuration usually responds rapidly to treatment, but in a depressing number relapse follows soon after the infant returns home. In these cases we have noticed rather a curious sequel. We readmit the child but withhold antibiotics and frequently, with simple aural toilet, the infection subsides and the ear becomes dry. We accept this with gratitude tinged with apprehension. Have we in fact succeeded in converting an overt suppuration into a closed mastoiditis? We have a number of these children under regular review and we hope to publish a long-term survey of their later history which may clarify this problem.

Where, however, the otorrhœa persists, we have found that a second course of antibiotics is likely to be as ineffective as the first. In other words, where relapse follows rapidly after a thorough course of antibiotics there is probably established disease within the mastoid. In such cases we do not hesitate to explore surgically. I am not prepared to say what the long-term results of this surgery will turn out to be, but I am quite certain that as an immediate policy it is entirely justifiable. Even in tiny children relapsing otorrhœa indicates irreversible mastoid pathology and calls for early exploration if the child is to be spared the terrible burden of chronic disease in later life. Even more so when acute otitis media supervenes in older children we should not lightly assume that it is an entirely new development. It may well be the final outburst of a disease which has been grumbling within the mastoid for a considerable period. Whether at operation the surgeon finds black cholesteatosis or true epidermosis, glutinous exudate or frank pus, depends on the particular course which the preceding disease has run. All cases, however, are to be regarded as examples of the same vast symptom complex under different guises.

Thus we have been compelled, much against our will, to resort to surgery with increasing frequency. Where the disease is localized to the mastoid air cells the immediate results are excellent. Where true epidermosis has occurred the technical difficulties are enormously increased and the results correspondingly poor.

The picture I have painted is a gloomy one—perhaps too gloomy. I will admit that in reviewing a series of these children we have frequently been gratified to observe a striking late improvement—the disease seems to burn itself out. Nevertheless the stigmata remain and bode ill for the

child's future; moreover it is impossible to exaggerate the amount of suffering and ill-health resulting from this widespread disease and there is certainly no room for complacency when we contemplate the incidence of its pyogenic complications. Above all I submit that it is wrong to regard it as a purely rhinological problem. If what I have said about the environmental and potentiating factors is acceptable it follows that the attack on this disease must be carried into the realms of public health on the broadest possible front and it is our duty as rhinologists to insist that this shall be done.

Acknowledgments.—The work on which this paper is based was carried out under the supervision and direction of my chief, Mr. A. Tumarkin, and owes much to his inspiration and encouragement. As head of the Department of Oto-Laryngology of Liverpool University he has generously placed at my disposal all facilities and I am glad to record my thanks to him and to the University. This work is the clinical aspect of a major research programme, the scope of which has been more fully outlined in his monograph "On the Nature and Vicissitudes of the Accessory Air Spaces of the Middle Ear" (Tumarkin, 1957). That programme enjoys the financial assistance of the Medical Research Council and I am happy to acknowledge the support which I have thus indirectly obtained from the Council. I am also glad to record my thanks to the staff of the John Bagot Hospital where much of the clinical work was done and to the Liverpool North Area Management Committee who have so consistently supported the project.

REFERENCES

CHANOCK, R. M., *et al.* (1958) *New Engl. J. Med.*, **258**, 207.
——, *et al.* (1959) *J. Amer. med. Ass.*, **169**, 548.
DIAMANT, M. (1940) *Acta otolaryng., Stockh.*, Suppl. **41**.
——, (1949) *Acta otolaryng., Stockh.*, **36**, 520.
FABRICANT, N. D. (1941) *Trans. Amer. Laryng. Ass.*, 231.
—— (1945) *Arch. Otolaryng., Chicago*, **41**, 53.
FLEMING, A. (1929) *Lancet*, i, 217.
FRY, J. (1958) *Brit. med. J.*, ii, 883.
GORHAM, E. (1958) *Lancet*, ii, 691.
LAWTHER, P. J. (1958) *Proc. R. Soc. Med.*, **51**, 263.
MASTERS, P. L., *et al.* (1958) *Brit. med. J.*, i, 1200.
Medical Research Council Survey (1957) *Lancet*, ii, 510.
MILLIGAN, W., and WINGRAVE, W. (1911) A Practical Handbook of the Diseases of the Ear. London.
NEGUS, V. (1958) Comparative Anatomy and Physiology of the Nose and Paranasal Sinuses. London.
OGILVIE, A. G., and NEWELL, D. J. (1957) Chronic Bronchitis in Newcastle upon Tyne. Edinburgh.
OSWALD, N. C. (1958) Recent Trends in Chronic Bronchitis. London.
PARKINSON, S. N. (1945) *Arch. Otolaryng., Chicago*, **41**, 68.
SENTURIA, B. H., *et al.* (1958) *Ann. Otol. Rhinol. Laryng.*, **67**, 440.
TUMARKIN, A. (1957) *J. Laryng.*, **71**, 65, 137, 211.
TWEEDIE, A. R. (1934) *J. Laryng.*, **49**, 586.

Mr. H. S. Sharp referred to cases of upper respiratory catarrh, occurring in childhood, which were basically allergic in origin but which only had their symptoms in the winter months. He called them the "winter wheezers", since in the summer time they were free from symptoms; the opening speaker had described them well. Usually they were in the age group 2 to 6 years and gave a classical picture of recurrent attacks of wheezy breathing, easily heard by the mother, a watery nasal discharge and a severe cough. The cough was chiefly troublesome at night, but could be annoying in the daytime as well, and was prone to be brought on by exercise. It was non-productive, harsh, and extremely persistent and noisy. The child might have frequent episodes each night, and though the cough was—or seemed to be—very severe, he might not himself wake up though he would probably wake everyone else in the house. These episodes were always brought on by an upper respiratory infection, which might be mild or severe, but which in these children triggered off the reaction of the allergic mucosa in their upper respiratory tract.

For the treatment of these winter wheezers ephedrine and promethazine hydrochloride were of supreme value. At Great Ormond Street it was the practice to put these children on full doses of these drugs all through the winter months. The dosage varied with the weight and size of the child, but was usually ½ gr. b.d. of ephedrine and 25 mg. of promethazine hydrochloride at night. The infective upper respiratory element which set up the attack was also eliminated as far as possible.

Many of the children had diseased tonsils and adenoids or antra as their infective element, and if these were eliminated their allergic attacks were greatly reduced, and with the use of ephedrine and promethazine hydrochloride they could be kept completely under control.

There was a definite tendency for the condition to abate at about 8 years of age, and those children had been treated as outlined above. This fact was a help in giving a prognosis to parents and indeed to the child's general practitioner, who naturally had thoughts of possible emphysema or bronchiectasis in later life if the condition persisted.

Another aspect of upper respiratory catarrh concerned the use of prophylactic antibiotics. There was a growing tendency to use sulpha drugs and antibiotics of various types, usually sulphadimidine or penicillin V by mouth, whenever coryza occurred, or even to feed the antibiotics daily in small doses throughout the winter. Was this practice to be encouraged? Was there any evidence of benefit in it or was it considered harmful, and if so, why?

The Medical Unit at Great Ormond Street investigated the subject in 1954; the findings are shown in Table I. 48 children of school age were taken, these children being known to suffer from recurrent upper respiratory infections; some were from Mr. Sharp's own waiting list for tonsils and adenoids. The winter of 1954–1955 was divided into two four-month periods, and a pharmaceutical firm provided two lots of tablets, one batch containing 0·5 gram of sulphadimidine, the other sweet calcium lactate. Both lots of tablets looked and tasted alike, being flavoured with liquorice. For the first four months 24 of the children were given an inert tablet daily while the other 24 were given the sulphadimidine tablet, and the results are seen in the first two columns. For the second four-month period, January to April 1955, the two groups of 24 children received the alternative tablets, i.e. the group who had previously had the inactive tablet were given the sulpha ones, and *vice versa;* the results are seen in the middle two columns. A summary of the experiment is shown in the last two columns. In the inert tablet series there were 60 episodes of upper respiratory infection, as compared to 25 while the sulpha tablets were being taken. School week absence was 80 for the inert group and 30 for the active group. General practitioner visits were 41 as compared to 25.

TABLE I.—EFFECT OF PROPHYLACTIC CHEMOTHERAPY ON THE INCIDENCE OF ACUTE RESPIRATORY INFECTIONS, ABSENCE FROM SCHOOL AND NUMBER OF VISITS BY GENERAL PRACTITIONER (Burke, 1956)

	Sept.–Dec. 1954		Jan.–April 1955		Total		χ^2 calculated on total
	Ca	S	Ca	S	Ca	S	
Acute tonsillitis	26	10	20	11	46	21	
Bronchitis and pneumonia	3	1	5	0	8	1	
Otitis media	2	0	4	3	6	3	
Total	31	11	29	14	60	25	13·6 ($P<0·001$)
Absence from school in weeks ..	37	13½	43	16½	80	30	21·83 ($P<0·001$)
No. of visits by general practitioner ..	20	12	21	13	41	25	3·41 ($P<0·1$)

Ca = calcium. S = sulphadimidine.

The numbers were too few for any definite conclusions, but were brought forward in the hope that Members might have conducted similar investigations on a larger scale.

REFERENCE

BURKE, J. B. (1956) *Brit. med. J.*, i, 539.

Mr. G. H. Bateman stressed the importance of environment as one of the factors causing this trouble. In 1939 he had a long waiting list for T's and A's at the Belgrave Hospital in Kennington. The hospital closed when children were evacuated from London in 1940 and reopened in 1945. He then started seeing patients who returned and requested operation after the six years' interval. Very often the story was that they had been well while evacuated, but their catarrhal symptoms recurred on return to London. He did not know whether the important factor was climatic, family life or housing conditions.

Meeting
May, 1, 1959

A DISCUSSION was held on **Destruction of the Pituitary for Carcinomatosis.**

The opening papers were **Destruction of the Pituitary in cases of Carcinomatosis Secondary to Mammary Carcinoma.**—Mr. G. H. BATEMAN; **Attempt at Ablation of the Pituitary Gland by Implantation of Radioactive Material.** — Dr. FRANK ELLIS; **Some Pathological Findings in Cases with Radioactive Pituitary Implants.**—Dr. D. R. OPPENHEIMER.

The meeting will be reported in the *Journal of Laryngology*.

Meeting
July 17, 1959

SECTION OF LARYNGOLOGY WITH SECTION OF OTOLOGY

COMBINED SUMMER MEETING HELD AT THE DERBYSHIRE ROYAL INFIRMARY, DERBY

LARYNGOLOGICAL SESSION

Chairman—R. L. FLETT, F.R.C.S.
(President of the Section of Laryngology)

DR. HANS VON LEDEN (Chicago) showed films demonstrating some physiological points with regard to the larynx, and commented thereon.

Following the films a Discussion was held on **Laryngeal Physiology** in which the following speakers took part—SIR VICTOR NEGUS, Mr. MAXWELL P. ELLIS, MR. F. C. W. CAPPS, and MR. R. L. FLETT.

DR. HANS VON LEDEN replied to the Discussion.

The meeting will be reported in the *Journal of Laryngology*.

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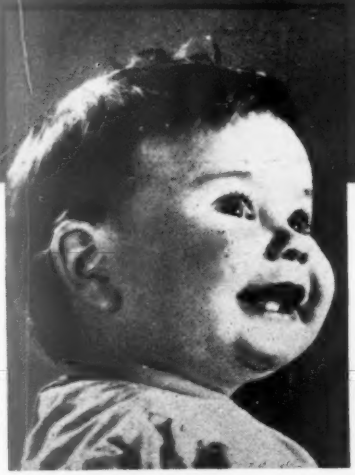

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Section of General Practice

President—G. J. V. CROSBY, C.B.E., M.D.

Meeting
April 15, 1959

DISCUSSION ON THE GENERAL PRACTITIONER IN THE MIDWIFERY SERVICE

Professor W. C. W. Nixon (London):

This discussion will bear particularly upon the recently published Report of the Cranbrook Committee on Maternity Services. Consideration of this Report has been occupying some of us very actively since its publication and doubtless many, like myself, have been examining and trying to weigh up the recommendations it makes for the improvement of our Maternity Services.

Improvement of social structures can be attempted in two ways, each having its own merits and drawbacks. Firstly, existing arrangements can be largely maintained, but modified and adjusted, and the danger of this is that the heavy weight of tradition may act as a drag; or secondly, a completely new arrangement can be devised, and here the danger is that much that is valuable may be lost. The Cranbrook Committee comes down on the side of making do with most of the existing organization. Its recommendations are regarded by some as being too timid, by others as having insufficient concern for their own interests. No one would regard them as final but, if even some of the proposed changes were attempted with real good will, and with a constant eye to the good of the whole, not only would the present situation be greatly improved, but the ground would be prepared in the best way for more radical changes in the future.

How can we best conceive an ideal maternity service? It seems to me we have to guide our thoughts in two ways. Firstly, we have, from time to time, to throw aside complexities or to pierce through them and try to form a clear picture of the biological needs of child-bearing women. Our vision will grow with experience and the real effort to comprehend. Secondly, without losing that vision, we have, as it were, to turn round and see how best those needs can be met amidst the complicated conditions of modern civilization. Each of you will vary a little in your concept of what, in the light of modern scientific knowledge, are the mother's essential physical requirements; you will vary more in your view of her psychological needs, but it seems to be accepted by all of you that, both in the physical and psychological care of child-bearing women, continuity is desirable. It was the lack of this continuity in the present arrangements which led to the setting up of the Cranbrook Committee in the first place.

In modern life the key to continuity of care is teamwork. This is an overworked word but we are a long way from having exhausted its possibilities or even of having comprehended its full meaning. It is clear that if the midwifery services are to be rescued from their present confusion it is imperative that we all learn to work together. There is no other way. In the case of the general practitioner obstetrician the person with whom he has to deal most closely, after the mother, is the midwife and something must be said about this relationship. It can be a very happy one. The Committee quotes the Report of the Working Party on Midwives (1949) who considered that the doctor and midwife should be partners "in the detection and treatment of abnormalities", but repeated the slogan that the midwife is "the practitioner of normal midwifery". Surely the time has come to drop that old battle cry? I have great sympathy with the midwives for they have had many hard and bitter fights. They are fearful that they will revert to an inferior status. We must do all we can to reassure them so that they do not regard the presence of a doctor at the delivery as any threat to their prestige whatsoever. Quite apart from his contribution to the welfare of the individual patient, the continued study of the natural history of labour is essential to the doctor; not only to enable him to gain a really intelligent understanding of the abnormalities with which he must deal but also to enable him to contribute to the better understanding (in which we will all share) of the causes of unnecessary abnormalities and of unnecessary pain and distress. For the doctor as well as the midwife has a special skill and training and therefore a special contribution to make. Perhaps I should say good doctors have. For there are bad doctors as well as good and another cause of difficulty in the past has been some doctors' ignorance of midwifery. It must indeed have been galling for a midwife to have to submit to the directions of one who knew so much less than herself and it must have made her

anxious for the welfare of her patient. Good teamwork needs trust and respect from both sides. To me it seems one of the biggest blots on the profession at the present time that the British Medical Association should try to maintain that midwifery can be learnt by an undergraduate in eight weeks. As an examiner and teacher of students I for one am not under the delusion that a man's ability to pass the Finals examination immediately gains for him specialized competence in all medical subjects. Who of us in this room would wish a near relative of our own to be in the hands of a newly qualified practitioner who had had no resident obstetric postgraduate experience? None, I am sure; and I know too that in speaking to you, who take the trouble to come to meetings like this, I am speaking to the converted. We must all stand for the dignity and honour of our profession and insist on some minimum satisfactory criteria for entering and remaining on the obstetric list.

The Committee begs the midwife not to be "possessive" about her patients and this injunction could apply to all of us (even the general practitioner obstetrician!). For example, the consultant might also be admitted to the team as a regular member, not merely invited in when difficulty has arisen. He may perhaps feel justified in thinking that his years of study and experience have enabled him to make a useful contribution. He cannot displace the family doctor from his special position. But the consultant should play his due part and assist in determining the common policy. Hospital obstetricians have often been at fault in not fostering more liaison between the hospitals and the general practitioners. Information certainly, but personnel also, might profitably be exchanged. Young registrars would gain immensely by being allowed some insight into domiciliary practice and general practitioners should be allowed to refresh their acquaintance with hospital work. I for one would very much like to encourage such interchange.

An important recommendation of the Committee is that, in close association with a hospital, beds should be provided for general practitioner obstetricians. I was impressed by the suggestion of Drs. J. Sluglett and Sarah Walker of Bristol of a "short stay general practitioner unit attached to a maternity hospital" in which the mother's own doctor and midwife would look after her during labour and after her early discharge home. (I would suggest within twenty-four hours.) The Committee thought that this scheme would raise administrative difficulties with catering, cleaning, sterilizing and so on, and I can see that this might be so. But surely it is not beyond the wit of man to solve difficulties of that sort in the interests of a scheme that, as a whole, represents progress, which I think the proposed one does.

Another matter of practical organization is concerned in the Committee's suggestion that the general practitioner obstetrician would more easily be able to do his work well if his general list were limited. At the same time it was thought that this arrangement would occur automatically if he were better paid. The point was also made that there are advantages in working in a group practice. It is not, of course, always possible. But I do know from my own experience in general practice how difficult it was to give adequate attention to a mother in labour if one had to struggle alone through a long evening's surgery. Another important practical consideration is the provision of facilities such as X-ray and blood examinations. However, decisions about responsibility for this will turn upon the shape and structure of the organization as a whole. It may, perhaps, still concern the Local Authority doctor, whose part in the scheme we must now consider.

On this point there seems to be very wide agreement: that the Local Authority doctor, who has done such splendid and pioneer work in the past, is not the right person to give antenatal care to patients for whose delivery he will have no responsibility. The Local Authority doctor, however, is especially skilled in preventive medicine and health education and is well equipped, as the Committee points out, to teach mothercraft and hygiene. He is *not* equipped to undertake the preparation for labour, which is quite another matter from health education, though they may overlap. By its own confession the Cranbrook Committee was not especially conversant with this subject and failed to grasp the significance or even the existence of this most important distinction. Unless the preparation for labour is conducted by the person who will be responsible for the labour (or by someone very closely associated with that person), the teaching quickly loses its meaning and vitality and not only fails to help the mother, but can sometimes even do harm.

There remain many valuable contributions which the Local Authority can make to the teamwork. They know the mother's home conditions and their suitability for confinement, and they are experienced in following up non-attenders at the antenatal clinics. The Committee suggests that they might continue to provide clinic premises. The general practitioner might use these premises to examine his patients in association with the midwife and, on some occasions, with a con-

sultant. In some localities it is more convenient for the midwife and doctor to see the patient in the surgery. In this, as in many other matters, organization must always be flexible enough to allow adaptation to varying local conditions and to allow for experimentation and individual initiative.

Finally there is the question of a co-ordinating committee. Many of us are tired of committees, but it is difficult to see how we can do without them in the complicated democratic society in which we live. A co-ordinating committee seems essential. Perhaps as well as discussing administration it could foster clinical meetings, of the sort in which there is free and informal exchange of views. Ultimately, it is upon good human relations that the creation of a wisely and humanely planned maternity service depends.

REFERENCE

Ministry of Health, Scotland. (1949) Report of the Working Party on Midwives. London.

Dr. Jean M. Mackintosh (Birmingham):

I do not agree with those who say that general practitioners have no part to play in the midwifery service, either in the domiciliary field or in hospital. While our medical qualification entitles us all to practise medicine, surgery and midwifery to the best of our ability, we are wise if we recognize our own limitations and concentrate our efforts on those fields of medicine where we are most likely to do our patients and ourselves justice. So, while all qualified medical practitioners are entitled to practise midwifery, because it is an art which requires special skill and experience which must be maintained, only those general practitioners with this special skill and experience should receive additional remuneration for their midwifery work.

Let us look at the facts as they are at the present time. How can the general practitioner obtain and maintain the necessary skill? There are those who argue strongly that if the training of the medical student in midwifery were more adequate than it is at the present time, all newly qualified medical practitioners would be adequately equipped to undertake domiciliary midwifery. It is very certain that the training medical students receive at the present time in many instances falls far short of this standard. It is not that their teachers are not alive to the situation, but the pressure of work in the medical curriculum makes it impossible for the student to be taught more than the fundamental principles of the subject and gives him very little experience in the practical application of these principles. It is for this reason that the Cranbrook Committee recommended that before admission to the obstetric list practitioners should have held a resident appointment in midwifery.

Yet even if it were possible to train the medical student, so that he was fully equipped to undertake midwifery when newly qualified, we are faced with the problem of how this special skill is to be maintained. Here, again, we must be realistic. Even witnesses opposed to the obstetric list agreed that, in order to remain competent, a doctor ought to attend a minimum number of cases each year. Experience has shown that, because of its size, what happens in Birmingham is fairly typical of what happens or can happen in the country as a whole, as far as the urban areas are concerned, and therefore a short account of the Birmingham situation may be of interest.

In Birmingham in 1957, which is the latest year on which I have detailed information, there were 18,714 live births and 415 stillbirths in the city to women resident in Birmingham, a total of 19,129 births. The number of infants born in hospital was 12,514, of whom 1,022 were born following emergency admission of their mothers. A proportion of these emergency admissions related to women requiring admission for social reasons, for whom a bed could not be found before the onset of labour. This left a total of 6,615 domiciliary births. The number of general practitioners doing domiciliary midwifery was 541. On that basis there was an average of 12 domiciliary births per general practitioner. Of course the domiciliary births are not evenly distributed among the general practitioners concerned. In all there were 303 practices undertaking midwifery. If the standard set by the Cranbrook Committee of an average of 20 cases per year over a period of three years is applied, then on the basis of their domiciliary practice in 1957, only 14% of the 143 single-handed practitioners would be qualified to remain on the obstetric list. In the 103 practices where there were two doctors, 46% had more than 20 midwifery patients during the year, but only 9% had more than 40 deliveries per annum. At the other end of the scale there were four large practices with four or five doctors working together, who had midwifery case loads of 84, 90, 129 and 153 patients respectively and whose numerical requirements, according to Cranbrook standards, were well covered for all the practitioners working in them.

In Birmingham, therefore, one-third of the 303 practices undertook sufficient domiciliary mid-

wifery to allow at least one of the partners or assistants to remain on the obstetric list. In 1957, 65% of Birmingham babies were born in hospital. If the proportion of babies born in hospital is raised to 70%, the amount of domiciliary work available to general practitioners will be correspondingly less. It would seem very obvious therefore that only a proportion of general practitioners can hope to maintain the necessary skill if their midwifery practice is confined to the domiciliary field.

If, however, the recommendation of the Cranbrook Committee that any additional beds provided should be in general practitioner units is implemented, then the general practitioners on the obstetric list will have additional opportunities to maintain their skill. The further recommendation by the Committee, that the provision by a general practitioner obstetrician of full antenatal care for hospital-booked patients might be taken into account for his retention on the obstetric list, would also help in this direction.

Doctors working in sparsely populated areas would have difficulty in meeting these requirements. This was appreciated by the Cranbrook Committee, who expressed the hope that the Medical Practices Committee would not appoint a new doctor to such an area who had not held a postgraduate resident obstetric appointment. The Committee also recommend that in the few special areas where there is an unusually small number of births and where the annual average of 20 complete booked cases would be difficult to achieve, a lower number might be accepted provided the practitioner attended a refresher course approved by a medical school at least once every five years.

To enable him to give of his best to his patients it is important that the general practitioner obstetrician should have a close association with the other two branches of the health service. The keen consultant obstetrician should take an active interest, not only in the hospital services, but also in the domiciliary midwifery services in the area of influence of that hospital. He and the general practitioners in the area should work together as partners in the obstetric team, caring for all women confined in the area whether at home or in hospital. The wise general practitioner, too, will take full advantage of the ancillary services of the local health authority. Practitioners understand and value the services of the domiciliary midwife, but are less ready, in some instances, to use the educational services which can be provided by the health visitor either at their own surgeries or at local authority clinics.

If the recommendations as a whole of the Cranbrook Committee are accepted, the standing of the general practitioner who is really interested and experienced in midwifery will be greatly enhanced. As far as his individual patient is concerned the Committee would place firmly and squarely on his shoulders the responsibility for seeing that adequate arrangements are made for the care of his patient, whether she be confined at home or in hospital. As far as the needs of the community are concerned, he should be able, working in the obstetric team, to play an active part in the planning of the maternity services of the area.

Finally, because there have been misunderstandings of the recommendations of the Cranbrook Committee, may I emphasize that the Committee do not say that a doctor not on the obstetric list should be debarred from practising midwifery. What they do say is that the ordinary training of every doctor gives him the legal entitlement to practise midwifery, but that, in so far as he undertakes it without special skill or experience, there would appear to be no reason why he should be remunerated for such maternity work any differently than for any other advice and treatment he can give as a general practitioner. It follows, therefore, that special fees for maternity work, which should be substantial, should be payable only in respect of the exercise of special skill as recognized by a doctor's inclusion on the obstetric list.

Dr. C. W. Walker (Cambridge):

Thirty years ago most confinements in this country were attended by midwives and general practitioners. To-day the majority of women in this country are confined in hospital, by midwives it is true, but by midwives under the supervision not of general practitioners but of consultants. With the help of Dr. C. G. Eastwood, Medical Officer of Health of the City of Cambridge, I have extracted some figures (summarized in Fig. 1) from the Annual Reports of the Medical Officers of Health of Cambridge 1930–1957 which illustrate the trend of events and are probably fairly typical of the country as a whole. In the period 1930–1935 20% of the parturient women of Cambridge were confined away from home, most of them in small private nursing homes. Even at that time many women liked being away from home. The nursing home was often owned by the midwife who would otherwise be doing domiciliary midwifery. The general practitioner found it convenient to have two or three patients under the same roof with a resident midwife. He would claim that his patient was safer. In the sense that

the convenience of the obstetrician is a factor in the safety of a woman in labour this was probably true, but the buildings and equipment of these small homes were not superior to the average council house. In this same period general practitioners cared for 96% of all confinements both normal and abnormal. From 1940 to 1945 confinements were nearly equally divided between hospitals, nursing homes and domiciles. Confidence in hospitals had been stimulated by the Midwives Act (1936) and the work of Leonard Colebrook (1936; Colebrook and Maxted, 1936) on the prevention of puerperal sepsis. After 1948 private nursing homes faded out so that by 1955 the maternity hospital, now a teaching hospital, looked after more than 80% of the confinements of Cambridge women. Since then the proportion has fallen rather steeply so that in 1957 61% of the women were looked after in hospital.

One good reason for the popularity of hospital confinements is the safety of the mother. The maternal mortality has been reduced to one-tenth of the figure in 1930. This is an astonishing achievement which is due to the work of the hospitals. To-day it is true to say that the safest place for a woman to be confined in this country is in a maternity hospital. These facts have led to conclusions some of which I am unable to accept.

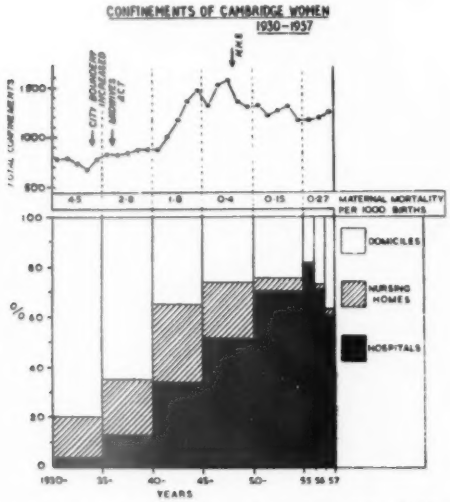

FIG. 1.—(1) Graph of total number of Cambridge women confined during 1930–1957. (2) Maternal Mortality per 1,000 viable births averaged for five-year periods. (3) Histogram of the percentage of domiciliary, nursing home and hospital confinements during the same five-year periods. N.B.—Almost all confinements outside hospital were the responsibility of general practitioners. The years 1955, 1956 and 1957 are not strictly comparable and are shown individually.

Some people, notably Fellows of the Royal College of Obstetricians and Gynæcologists, conclude that since the hospital is now the safest place for a woman's confinement this must always be so. The history of obstetrics shows that a woman in labour has not always been most safe in hospital. Furthermore, it is true that Sweden and Australia, where hospital confinement is the rule, have very low figures for maternal mortality, but so has Holland where only 22% are confined in hospital (Cranbrook Committee, 1959, p. 13). The question is important because if the conclusion of my learned colleagues is right we must obviously plan to have enough hospital beds for all women in labour. It is then impossible to accept the recommendation of the Cranbrook Committee (1959, p. 19, para. 57) that hospital beds should be provided for only 70% of confinements for they assume that, even with the most careful selection, 1–2% of those confined at home will become acute emergencies. If these women are not in fact receiving such a good service as they would receive in hospital, it means that every year 200 women may die in childbirth for lack of proper attention. No one expects plans to work perfectly in practice, but to plan the neglect of these women is intolerable. Before he accepts the idea of any home confinements, every general practitioner must so organize his obstetrics that every woman under his care is at least as safe as she would be in hospital. Is this really impossible?

It is necessary to analyse precisely the needs of a woman in pregnancy and labour and to discover the advantages in hospital confinement and whether these advantages are peculiar to hospital. A pregnant woman needs good antenatal care, the prevention and treatment of sepsis, shock and hæmorrhage in her labour and proper postnatal attention for herself and child. The hospital provides all this. How far is it possible to provide the same standards in the woman's home? It must be admitted that hospitals gave us a lead in antenatal care and recently the Ministry of Health (1955) has issued a memorandum advising both hospitals and general practitioners of the need for even more careful antenatal care. With proper co-ordination between general practitioner and hospital, it would seem that the pregnant woman who has booked her own doctor is not necessarily worse off than if she had booked into hospital. If during the pregnancy a complication of labour is foreseen, such as the probable need for Cæsarean section, the woman is recommended to book into hospital for her own safety because of the facilities obtainable in hospital. This really means that it is more facile, easy, convenient to make her safe in hospital. It is much more convenient to operate in hospital, but if time and money were

no object it would be possible for the consultant to import to the home his equipment, his staff and even his operating theatre should he so desire. In this way the patient could be made as safe in her own home as in hospital. All this is quite possible but highly inconvenient and extravagant. For our convenience we carefully select those patients who need, or are likely to need, continuous supervision and major operations for confinement in hospital. If general practitioner and midwife are willing and able to inconvenience themselves so that the patient gets the same attention as she would in hospital, there is no reason why quite serious abnormalities may not be supervised at home. With adequate transport and a reasonably accessible hospital, it is quite simple to transfer the patient to hospital if it is found she would be more conveniently delivered there.

Bacteriology, antiseptics and antibiotics are as readily available to the general practitioner as to the hospital, so that there is no reason why a woman should not be as well protected against sepsis in her own home as in hospital. It is relevant to note at this point the findings of Forfar and Maccabe (1958) and the report of the Nuffield Provincial Hospitals Trust on Sterilization in Hospital (1958).

The last great need of a woman in labour is the prevention and treatment of hæmorrhage and shock. As no one can foretell that a particular woman will not bleed, hæmorrhage is the commonest cause of the 1–2% of domiciliary confinements which become acute emergencies. The obstetrician whether in hospital or general practice will try to prevent postpartum hæmorrhage by carefully watching the labour and perhaps by using ergometrine during the second stage. If hæmorrhage occurs, the basic treatment is to stop the bleeding, empty the uterus and give an intravenous transfusion. It is therefore necessary for the general practitioner to be able to do bimanual compression, manual removal of placenta and put up a drip. He will carry transfusion-giving sets with plasma or dextran to every confinement. Just as the resident houseman will send for the registrar, so the general practitioner will send for the "flying squad" or consultant. In either case a sample of the patient's blood will be sent to the blood transfusion centre for cross matching. Nowadays, cross matching takes two hours in the laboratory, whether the blood comes from hospital or home. It would seem that the woman receives the same care for hæmorrhage whether in hospital or at home. It might be argued that an emergency in hospital is more quickly treated because there is always someone available. This is more true in theory than in practice because the general practitioner who attends his own confinements will be on the spot while the resident houseman may be engaged at some other operation.

Proper co-operation between general practitioner and hospital, as advocated by the Cranbrook Committee (1959, p. 83, para. 299), means that every patient who is to be confined in hospital should be seen from time to time by her own doctor. Probably it means that every patient to be confined by her own doctor should be seen at least once at hospital. The advantage of hospital confinements seems to lie not in buildings or equipment but in the differences in training and personality between houseman and general practitioner. It would be a serious fault in the training of a doctor and in the organization of the midwifery services if a man who had been trained to deal with emergencies in hospital were unable to deal with the same emergencies in the home, for lack of equipment or adequate help. The answer to our question is that it is possible, but not always convenient, to provide the same standards in the woman's home as in hospital. Obviously we shall suit our convenience to make the woman safe by referring to hospital those patients who are likely to cause trouble. At the same time, we shall take the trouble to provide for the emergencies which we cannot foretell in a particular patient by making provision in all cases.

Once it is recognized that women are booked into hospital because there they can conveniently be made safe, the selection of patients for hospital confinement becomes a matter of common sense rather than of regulations. Many women feel that a new baby is a family event and that the family should take part in this event and welcome the newcomer from the start. They, therefore, prefer to be confined at home. Some of them believe that labour itself is an exciting experience and they prefer to be at home under the care of the midwife and their own doctor and to be allowed to indulge to a certain extent their own desires about having or not having analgesics and drugs. Some take pride in showing their doctor how well they can manage their own labour. Normal labour deserves study so that what is at present fairly common may become general. Grantly Dick-Read (1947), a general practitioner, has given us a description of normal labour which is far in advance of anything which appears in textbooks. There are many unsolved problems which the field work of general practitioners may help to solve. It should be easier to do this field work in a patient's home than in hospital. At the same time, the practitioner must keep his knowledge and technique up to date and for this reason I welcome the suggestion that there

should be more general practitioner beds attached to maternity hospitals. In Cambridge, some general practitioners have signed on as post-graduate students and are allowed to attend a certain number of their own patients in hospital. These patients lie in the general ward and are not separated from the hospital patients. The practitioners have agreed to accept certain rules but are allowed considerable latitude in the treatment of their own patients and work in full view of the hospital staff. In this way they gain experience of hospital techniques and feel bound to keep to a high standard.

General practitioner obstetricians should be as far as possible self-selected, but no one who is not really keen should practise midwifery. Hadfield (1953) found that all general practitioners could be divided into three nearly equal groups: (1) those who were not interested in midwifery; (2) those who were really interested; (3) those who practised midwifery in order to keep the pot boiling. Ideally only group (2) should be general practitioner obstetricians. This would mean 6,000–7,000 instead of 14,000 as at present. If we assume that the total number of births in this country is 600,000 each year and that the new hospital beds for general practitioners will increase the proportion of maternity cases attended by general practitioners from 30 to 33%, then the number of cases available for general practitioners will be about 200,000. If there are 7,000 general practitioner obstetricians, each one will probably attend between 25 and 35 confinements a year. The minimum of 20 cases annually suggested by the Cranbrook Committee (1959, p. 55, para. 196) is so near the average that the number of obstetricians would tend to fall and there might be a dangerous scarcity in many areas.

At this point it is worth while noticing the flexibility of a general practitioner service. When births are few no money is spent on paying practitioners for standing idle. When births are numerous general practitioners can absorb the excess without the necessity to build more hospitals. This flexibility, if combined with an efficient hospital co-operation, will save the country a lot of money without endangering the mothers.

Properly done, obstetrics is a physical strain and time-consuming. Such work is suitable for younger men and women. If they are keen to do the work, they will take the necessary training in hospital jobs if such jobs are available. New applicants to the obstetric list should not find it difficult to fulfil the criteria for admission advocated by the Cranbrook Committee (1959, p. 55, para. 195). On the other hand, the review every three years might exclude young practitioners who had not been able to build up an obstetric practice. It might also exclude older and more experienced practitioners who wanted to cut down their midwifery. The suggestions about payment (Cranbrook Committee, 1959, p. 56, para. 206) appear to be unworkable without an Act of Parliament and probably unprofitable if an Act were passed.

The value of experience in general practitioner obstetrics is difficult to assess. The question is "Experience in what?" The young practitioner anxious to gain experience in spontaneous delivery might expect to achieve this if he conscientiously attended 20 booked cases a year. On the other hand, if he wished to gain experience in the application of forceps, he might still achieve his aim with 20 cases a year, to the detriment, some might think, of the highest standards of midwifery. The best way to gain experience in a novel situation or unpractised technique is to ask the help of a colleague, either general practitioner or consultant. However stiff the criteria for the obstetric list, there are bound to be gaps in the practitioner's experience. The danger is that the young doctor, proud of his achievement in getting on the list and anxious to gain experience in what he knows he lacks, should buy his experience too dearly. To me it is much more important that help should be readily available than that all general practitioner obstetricians should be experienced. The emergencies of midwifery are not so common that any obstetrician can expect to gain experience in them except after years of practice. He must, however, be prepared to diagnose an acute emergency and to take appropriate first-aid action. In this I would include manual removal of the placenta and the treatment of hæmorrhage, if necessary with intravenous transfusion. He may possibly gain experience in manual removal and probably in the treatment of hæmorrhage in hospital, but he will get little experience in the early diagnosis of an emergency. This is a lesson which is bitterly learnt in general practice.

What we need on the obstetric list is a body of men and women, many quite young, who are able to make observations on normal and abnormal labour. These obstetricians will have such effective help and techniques at their disposal that women in childbirth will lose their fear and be relaxed and natural. The slick but obtrusive efficiency of the hospital will give place in the home to a form of care, which is so well hidden that it never troubles the mind of the mother and allows nature free, but carefully watched, play. There is a real danger that motherhood, an exciting event in the life of the family, will become an unpleasant episode, like the removal of an

inflamed appendix, which interrupts family life and is best forgotten. The general practitioner is in a unique position to prevent this. He can watch the natural unfolding of the processes of pregnancy and labour, but be ready to interfere, not only with his own resources but with all the resources of obstetric science, to prevent injury to mother or child when nature is failing. This is not an easy task. He will need the willing help of midwives, consultants and hospitals, as well as the confidence of his patient, if he is to succeed. If those who are on our obstetric lists are keen on the job, the task, though difficult, is not impossible.

REFERENCES

Annual Reports of the Medical Officers of Health for the Borough (City) of Cambridge, 1930–1957.

COLEBROOK, L. (1936) The Prevention of Puerperal Sepsis. Manchester.

——, and MAXTED, W. R. (1936) Antisepsis in Midwifery. Manchester.

Cranbrook Committee (1959) Report of the Maternity Services Committee, Ministry of Health. London.

DICK-READ, G. (1947) The Birth of a Child. London.

FORFAR, J. O., and MACCABE, A. F. (1958) *Brit. med. J.*, i, 76.

HADFIELD, S. J. (1953) *Brit. med. J.*, ii, 692.

Ministry of Health (1955) Antenatal Care Related to Toxæmia. Memorandum, Central Health Services Council, Standing Maternity and Midwifery Advisory Committee, London.

Nuffield Provincial Hospitals Trust (1958) Present Sterilizing Practice in Six Hospitals. London.

Miss A. Wood (General Secretary, Royal College of Midwives, London):

The General Practitioner in the Midwifery Services
The Point of View of the Midwife

"We are convinced that it is desirable that a midwife should be given every opportunity to participate in the maternity care of her patients to the fullest extent to which her skill and experience entitles her." This statement in the Report of the Cranbrook Committee (para. 107), while appearing to give the midwife her full share of responsibility, can be interpreted in different ways. For instance, the words "fullest extent" may mean one thing to the midwife and another to the general practitioner, and still another to the consultant obstetrician. One wonders what the members of the Cranbrook Committee had in mind when they wrote them. They do not elaborate the statement, but I think it must be read in the light of their comments on the Report of the Working Party on Midwives. They lay great stress on the value to her patients of the midwife's "assets of time, skill and attitude of mind", but they do not say that they agree with the Working Party's statement that the midwife should be "the practitioner of normal midwifery, the expert in all its aspects". Neither do they say that they disagree with it, but in other parts of the Report the Committee make it clear that they think the general practitioner should play a bigger part in normal midwifery than he does at present. Whereas the Working Party Report stated that the doctor was the partner of the midwife "in the detection and treatment of abnormalities", the Cranbrook Committee seem to regard them as partners in more than this, for they say that all necessary maternity care should be shared between them.

The Central Midwives Board have said that the training of the midwife in Great Britain aims to produce a woman who "is adequately trained in both institutional and domiciliary practice, capable of conducting normal deliveries on her own responsibility, of giving in normal cases the requisite advice and attention in both the antenatal and postnatal periods, and of recognizing the circumstances in which the attention of the doctor is necessary". This means that during her training the midwife has acquired not only the skill to manage a normal labour and deliver a woman of her child, but also the many other skills which are necessary for the proper care of the mother during the whole of her pregnancy, in the postnatal period and also for the management of the newborn baby. In addition to this practical part of her work, the midwife has an important part to play in health education. She is the appropriate person to teach the mother the physiology of labour, the preparations to make for it and the use of analgesic apparatus. The teaching of mothercraft is now included in the syllabus of the pupil-midwife and the midwife should advise the mother about breast-feeding, how to prepare for it, what to get ready for the baby, and how to care for him when he is born. Midwives can also teach small groups of mothers relaxation and antenatal exercises, and they must be able to assess the suitability of the home for confinement. The Cranbrook Committee have recommended that health education should be available to all expectant mothers and that Local Health Authorities should provide special "instructors" to go to doctors' surgeries and hospital antenatal clinics. They do not specify who these people should be, but we may assume that the Committee expect midwives and health visitors to co-operate closely in this work.

The midwife's asset of time is of great value to the expectant mother, as she can make herself available to discuss problems with her and to listen to what she has to say. When labour begins

the midwife again is the person who has the time to stay with the mother and she must be capable of making up her own mind about the progress of labour and of deciding when pain-relieving drugs should be given. Because the midwife is skilled and experienced in the normal, she is the better able to detect the first signs of the abnormal.

It is very important that we should bear in mind, when considering the relationship between doctor and midwife, what the midwife is trained to do. If the best care is to be given to the mothers and babies, it is obvious that this relationship must be a harmonious one, that the responsibilities of both doctor and midwife should be clearly defined and that both should obtain the maximum satisfaction from their work.

The Cranbrook Committee have said that in their view the doctor should be responsible for the co-ordination of maternity care, that wherever possible antenatal examinations should be carried out in the presence of both doctor and midwife, and that where possible the doctor should be present at the delivery, though he should not invariably undertake it. At the same time, the Committee do not wish to lessen the midwife's responsibilities and they have made the very sensible recommendation that the term "maternity nurse" should not be applied to qualified midwives, except those who notify their intention to practise as maternity nurses only. I am sure this recommendation will be welcomed by midwives and will help to convince them that their skill and experience as midwives are needed and valued by the doctors and that they do not wish to work with less well-qualified people. If this is in fact the case, it will be very important for the doctors to see that the midwives do carry their full share of responsibility, that they have opportunities to exercise their clinical judgment, to make up their own minds about a patient, and are not turned into maternity nurses who only act on a doctor's instructions. This may well happen, particularly with the younger generation of midwives, unless the doctors remind themselves of what the midwives are trained to do and allow them sufficient scope to practise their skills. The sharing of antenatal care should not mean that the midwife merely acts as the doctor's clerk or receptionist, but that she really feels responsible with him for the safety of the mothers they are looking after together.

It is estimated that some 80% of all deliveries are conducted by midwives and I think this is a record of which they may well be proud. But will this percentage be much reduced as a result of the Cranbrook Committee's recommendation that the general practitioner obstetrician should be present at the delivery whenever possible, and that in order to remain on the obstetric list he must have attended at least 10 deliveries a year? This number does not sound very high but much will depend on how many general practitioner obstetricians there are and whether they will be content to leave the actual delivery to the midwife while they help the mother with the gas and air or Trilene machine.

I feel sure midwives will welcome the recommendation of the Cranbrook Committee as to the criteria necessary for a general practitioner to be placed on the obstetric list, because this should substantially improve the standard of maternity medical care. Midwives have been accused of being over-possessive and anxious to keep the realms of normal midwifery in their own hands. They have perhaps clung too rigidly to the dictum that they are "the practitioners of normal midwifery", but I think there is some justification for the fear that has existed, and still exists with some of them, that the practice of normal midwifery is gradually passing into the hands of the doctors and that there will soon be no point in taking the second part of the midwifery training. I think that general practitioners and obstetricians can do a great deal to dispel any fears of this kind by giving hospital and domiciliary midwives as much responsibility as they can, but I also think that midwives must show themselves willing and able to take the responsibility. They should also realize that doctors often feel the need of more experience in normal midwifery, that they are interested in the mothers they look after and dislike being called in at the last moment when things have begun to go wrong. Both doctor and midwife have a great deal to contribute to the care of the mother and baby during pregnancy, labour and the postnatal period and the important thing is for each of them to recognize this fact, whether they are working in hospital or on the district, and to see that they understand quite clearly what are their individual responsibilities.

The Cranbrook Committee have been criticized by some people for not having recommended any change in the tripartite administration of the maternity services. It seems to me that their reasons for not doing so were sound and that they were wise to concentrate on suggestions for closer co-ordination of the three parts of the service. One difficulty which faced the Committee, when they were considering the possibility of the domiciliary midwifery service being taken over by the hospitals, was that the domiciliary midwife is very often the district nurse as well and sometimes also the health visitor. It would indeed lead to complications if she became an employee of two different authorities. But this does not mean that there cannot be a closer liaison between

hospital and domiciliary midwives. For instance, in urban areas maternity hospitals or units might provide domiciliary midwives for the district immediately surrounding the hospital. These midwives would be part of the hospital staff and they could help in the hospital from time to time, if the district were slack and the hospital busy. There is of course nothing new in this suggestion and it is already done very successfully in many places. But it could be extended and it certainly has many advantages, both from the point of view of the mother and of the midwives. The recommendations of the Cranbrook Committee for better co-ordination of the maternity services are sensible and practical. Standard co-operation cards should prove extremely useful in ensuring full exchange of information, and the appointment of local liaison committees, composed of professional people, should bring the three parts of the service closer together, provided of course that the members of the Committee can agree on a common policy.

I hope that hospital and domiciliary midwives will take their full share of responsibility for co-ordination and co-operation as members of these committees. I think also that their attendance at the local clinical meetings could be extremely helpful. In the last resort, co-operation can only take place between individuals and if their relationship is right it surely does not matter very much if they are employed by different authorities. If the needs of the mothers and babies are kept uppermost in their minds, they will not go far wrong and it should not be necessary to make elaborate administrative arrangements to ensure real co-operation.

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Meeting
December 12, 1957

The Influence of Eye Disease on Pictorial Art

By P. D. TREVOR-ROPER, M.D., F.R.C.S.
London

TOWARDS the end of last century, the mysteries of biology were, one by one, becoming reduced to a simple organic explanation, and on the crest of this wave of iconoclasm, religion and the fine arts seemed ready victims. So in turn we find the revered paintings of the past being blithely dismissed as the by-products of a miscellany of eye diseases—astigmatism, cataract, colour-blindness and so on. Nowadays these rather frail mechanistic interpretations have gone out of fashion as psychology has "come in", and from this new and comfortable pastime of nibbling away at the artist's ego, no one has much time for those crude old organic interpretations. But the trouble is that one cannot dismiss them quite as easily as all that; so with all the proper humility of a simple technician I would like to lead you through the evidence, such as it is; and in rash moments fly a few small kites—remembering always that what I say can be only fairly applied to naturalistic paintings, which have been of quite secondary concern in most periods and most cultures.

THE REFRACTIVE ERRORS

Astigmatism

First of all the eye disease responsible for the most famous and the least convincing of all these theories—astigmatism.

Since the eyeball is rarely, if ever, an exact sphere, a little flattening in any meridian will inevitably reduce the height of the retinal image in that meridian, making it seem disproportionately broad[1] (Fig. 1); and in the same way, the image perceived by the brain, which crudely corresponds to the retinal image, will be broader too. Astigmats then see objects as broader or as taller than they really are if their eyeballs are a little flattened from above downwards or sideways.

Astigmatism is an almost universal disease, and although it was only in 1825 that Sir George Airy fashioned the first correcting spectacle-lens, fifty years earlier a German high-school teacher had indeed declared "experience tells me that I have a somewhat exaggerated impression of the object in comparison with its height" (Fischer, 1783).

El Greco is the classically quoted instance, for in nearly all his paintings there is a vertical elongation, but with an added obliquity which makes all his characters seem to be in danger of sliding off the bottom right-hand corner of the picture.

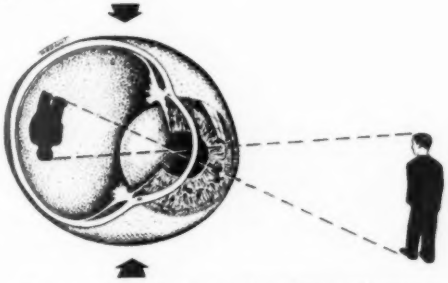

FIG. 1.—The effect of astigmatism. Diagram to show how the retinal image is broadened when the eyeball is flattened from above downwards.

This astigmatism of El Greco's seems to have first been suggested in the Paris Medical Chronicle of 1913; thereafter came a desultory and generally sceptical discussion, mostly in the German and Spanish ophthalmic literature (Greeff, 1915; Patry, 1917; Levi-Sander, 1917; Isakowitz, 1918, 1933; Marquez, 1926, 1929; Huber, 1932, 1935*b*; Villa Ortiz, 1934), and even in recent years this theory has not lacked occasional advocates (Ahlström, 1955; Bozzoli, 1957).

The primary objection has always been securely based on the historical setting of El Greco's work, with his Cretan compromise between a Venetian naturalism and perspective, and an underlying traditional Byzantine stylization; and this is cogently supported by the modern evidence from X-rays (showing that the elongations were secondarily imposed on the original sketches). But these authors seemed more concerned with various incidental points,

[1]In point of fact the actual disproportion is very slight—about 1% in a moderate astigmat (Le Grand, 1952).

such as the occasional elongation of features, especially hands, when these lay horizontally (as in the portrait of Cardinal Tavera), and the relatively normally shaped faces of the figures in the foreground of the painting of Toledo (possibly including the donor of the portrait who required a more naturalistic conception of himself); and this last point led several of the protagonists to argue that El Greco's astigmatism forced him to elongate only when he was drawing imaginary figures—without a "sitter" to portray, or that it obtained only in astigmatism which was "acquired" late in life as opposed to the normal congenital form.

In point of fact the likeliest mechanical explanation has somehow been overlooked, for this elongation towards the bottom right-hand corner of the canvas is a natural tendency among right-handed artists, who, watching their subjects across the top left-hand corner, find the bottom right-hand corner receding tangentially from their orbit of projection.

This familiar elongation of El Greco's is typically seen in the portrait of the Cardinal Inquisitor Nino de Guevara (Fig. 2), which has an incidental ophthalmological interest in that the Cardinal is wearing a pair of archetypal spectacles, fastened with a cord behind the ears in the Chinese fashion. On the left-hand side of Fig. 2 is the original; while the right-hand picture is a photograph of it through a $-1{\cdot}0$ D. astigmatic lens at 15° axis, taken by Ahlström using an anamorphic lens which had this resultant power; and it is undeniable that this lens has largely restored the correct proportions, as well as removing the rather disquieting malequilibrium of the Cardinal, who had seemed to be slipping off his chair.

However, the various instances of purely horizontal and vertical elongations, which have been attributed to an eyeball flattened from above downwards, or sideways, are rather less familiar. In the well-remembered rendering by Hans Holbein the Younger of King Henry VIII (Fig. 3A), as in many of Holbein's other portraits, the subject is broadened, presumably to make him look more portentous; and again, Fig. 3B shows Ahlström's reduction of its width by photographing it through the same $-1{\cdot}0$ D. astigmatic lens, but this time on a vertical axis, from which it has been suggested that Holbein was astigmatic with a vertically-compressed eye (as was illustrated diagrammatically in Fig. 1). Are we so sure that this too is a stylization when we discover that in portraying his figures recumbent, as in his "Dead Body of Christ" (Fig. 4), Holbein generally makes them long and thin, not broad and fat?—all this in spite of the various mechanical aids with which Holbein sought to reproduce the proper dimensions of his subject.

The opposite axis of elongation can be illustrated by Lucas Cranach "the elder" (Fig. 5); but with these vertical elongations the astigmatic theory is far more unconvincing (since fashion often favours a spindly figure). Nevertheless even Botticelli has been labelled an astigmat (Huber, 1932; Cantamessa, 1938) and so has Titian (Huber, 1932), not to mention even more striking elongators like Modigliani. All these suggestions, of course, admit little serious consideration, quite apart from the basic counter-evidence that Modigliani's recumbent nudes and Botticelli's horizontally stretched hands are just as elongated as those that are upright.

However, by the time we reach John Singer Sargent, we do apparently have a known astigmat, and as a result it is reported that he used to see a red or green line around white objects which he often painted as in fact (Mills, 1936). Finally there is our great contemporary, F. B., who has a marked horizontal astigmatism in each eye. Before the war he used to paint without glasses, but then found that when he examined these paintings with his correcting lenses they all seemed too elongated, and the brush strokes seemed too coarse and broken-up, with the result that the images appeared to have lost all their compactness. He hunted for some of these early paintings so that we could contrast them with their "corrected" equivalents, but I am afraid in vain, and I can only quote those impromptu comments that he made. And there is also an astigmatic Dublin Academician (P. H.) who volunteered that he always tended to draw his vertical lines slightly oblique, since truly vertical lines tended to "shimmer", by which he meant that they were blurred like the spokes of the sight-tester's "astigmatic fan". Many artists and art-teachers have noted this difficulty of drawing true verticals and horizontals, although most of them have quick recourse to a mirror and erase this evidence of their (?) astigmatism.

Before we start to consider this theory, that the shape of the painted image is distorted in proportion to the artist's astigmatism, we must face the cardinal objection (which will apply in a different measure to cataract and the rest) that the artist paints what he sees, and the subject will correspond to the rendering, however much they are both altered by the misshapen eye into a distorted percept within the artist's occipital lobe. In other words that if he sees a flattened or elongated world, the likeness of it that he puts on to the canvas, in order to appear equally flattened or elongated to him, will in fact be depicted with its proper dimensions. However, we shall later see that

there is just enough evidence that this self-regulating effect does not invariably apply, for this theory to merit our patient, if sceptical, consideration.

Myopia and Hypermetropia

Of the next two refractive errors, myopia or short-sightedness implies an eyeball which is simply too long, like a folding camera that is pulled out too far, and so focused only on near objects, and hypermetropia or long-sightedness implies a short eyeball which has correspondingly less ease at focusing near objects.

The *myopic* (or short-sighted) painter can thus clearly see the canvas, but not the more distant object he is painting, and he therefore is reduced to painting what he sees, however blurred or distorted a percept it is. Beyond the farthest point of his natural focus, vision becomes increasingly a sort of "peripheral vision" such as the normal-sighted person sees out of the corner of his eye, with a loss of detail and with a relative clarity only of the essential lines and contours. And to illustrate this let me quote the following graphic description by Mills (1936), of what the myope, and particularly one with an added astigmatism, actually sees. The farthest point of his clear vision is only about 15 cm. away; within this range, he says,

> "I appreciate fine and almost microscopic detail; but beyond this, and especially at distances over 6 metres, objects become greatly blurred and colours run together with curious blends and unusual, washed-out values. There is definite oblique distortion at far distances, differing in the two eyes, and often only the essential lines of form and contour provide the clues for identification of the object under examination. Such lines frequently take the jazz mathematical shapes of cubism, and if I were a painter my conception often would be essentially geometric. At the symphony concerts my seat is in about the centre of the pit, nearly 70 feet from the stage. Three points of attention fix my interest at once: the tall form of the leader in the centre, attenuated like an El Greco drawing, two golden harps on the left flank and a strong white reflection from the curved, glistening, light-brown barrel of the bass drum, all striving for attention. The conductor holds the centre of interest, gyrating in strange contortions like some fearful wizard before a medley of misshapen geometric patterns in blacks, greys, whites and brown and gold; there are no details anywhere, merely blurred outlines of colour, form, light and movement. The cellos, on the right, appear like enormous yellow-brown gourds placed on the bias; the hands of the cellists form curious patterns, the bowing hand being a yellowish disk weaving in and out, while the hand which weaves over the frets plays up and down at a wholly different angle of inclination. The white shirts of the drummer and of the two men on the extreme flank of the bass viols appear as blots of white and all other shirts as vertical slits. The faces of the players of the stringed instruments form a veritable flower pattern, like great nodding daisies, in whites and yellow-browns; the bowing hands weave up and down as yellowish disks. The black clothes of the conductor and of the row of men next to the audience, that is, the men farthest from the strong overhead illumination, are jet-black, while the identical apparel of the rest of the musicians, directly under the lights, is grey-black, the contrast being sharp. When the harps are seen with one eye and then with the other eye, there is a prompt change in the angle of their inclination from the vertical, which represents the difference in slant given by the different degrees of astigmatism in the two eyes."[1]

This "peripheral" type of imagery is, of course, familiar to us, but rarely analysed (Fig. 6 is an attempt to illustrate the loss of acuity even close to the fixation point), while for those who are short-sighted, it is the sort of view always obtained without their glasses; and it is that made use of by artists who aim primarily for effects of mass, line, colour and symbolism, just as it can be used by the lazy or the immature (as in primitive or child-art); while it has been triumphantly exploited in the hands of the impressionist school.

We can only guess to what extent Monet, who so pioneered the cultivation of this peripheral type of vision, was myopic (as well as having a cataract); but we know Cézanne was, even without the indirect evidence from his paintings (such as Fig. 29), and it is not surprising that only in some of his self-portraits are his colour values and optical proportions at all conventional. He incidentally suffered also from diabetes, so who knows but that a little retinopathy may have further disturbed what Huysmans called his "diseased retinæ". Cézanne's distortions do indeed illustrate very beautifully his use of his own peripheral field[2] (however much they have been thoughtlessly stereotyped by his decorative imitators); and we know that he used to stare at his models for hours on end, before he dared to put down his first brush-stroke, liking them to sit motionless "like apples". As

[1] An Australian psychologist (J. R.) has just written to me, describing how she happened to look at the orchestra through the lower part of her bifocal glasses—rendering herself artificially myopic—and the scene "broke up into a remarkable and exciting 'modern' composition of triangles and planes and colour". And she is now trying to find out if some "modern" artists could throw their eyes out of focus at will and see actually—as she did—as an abstract painting (apparently with some success).

[2] Blanshard (1949) suggested that Cézanne actually saw the distortions of form in that immediate way in which previously the impressionists had actually seen the distortions of colour.

Ehrenzweig (1953) submits, "most of Cézanne's distortions somehow appear 'correct' if you do not try to see his shapes statically by a single glance, but abandon yourself to the movement inherent to his distortions"; the eye is then "led along definite highways to definite fixation points, and from this movement the whole picture will suddenly seem 'right'." His distortions are thus the product of a "realistic" vision, which took into account the eye's unconsciously guided movements that the static realism of his forerunners had neglected. And his myopia could well have provided an incentive or "short-cut" to his especial awareness of the true shapes and hues in the peripheral field. Then there was Renoir who was said by his biographer Vollard (1919) "to step back a few paces [in other words out of his limited near-range of clear vision], in order to give the painting the effect of an Impressionistic picture"; he was then 60: and even at 64, when none of us who is not myopic can hope to read at near range without convex spectacles, he describes how Renoir liked to examine petit-point close-to, taking it in his hands, and we know that he wore no glasses.

Some impressionists, like Degas, were highly myopic, for he wore heavy lenses throughout his adult life[1]; as a result he was reduced to painting in pastel rather than oil as being an easier medium for his failing sight; later he discovered that by using photographs of the models or horses he wished to depict, he was able to bring these comfortably within his limited focal range; and finally he fell back increasingly on sculpture where at least he could be sure that his haptic sense would always remain true. Among these myopic impressionists, it is tempting to throw in Pisarro, whose central vision was further impaired from the scars of corneal ulcers (possibly phlyctenular in origin) that had plagued him since he was 8 years old; not to mention others to whom myopia has also been imputed (Mills, 1936), like Dufy, Derain, Braque, and particularly Matisse (Fig. 7). Certainly the lack of perspective and depth of the Polish painter Matejko is excusably attributed to his myopia (Majewski, 1936), for his spectacles, bearing their tell-tale -4 D and -6 D lenses, have been preserved in the Krakow museum.

And lastly in this group we can perhaps include Gordon Craig, who was indeed so myopic that Isadora Duncan is said to have complained that he even failed to recognize her across the breakfast table! His biographer (Leeper, 1948) describes how he "always loved greys and browns, very low in tone" (as in Fig. 30), and the other myopic legacy—the emphasis of structure and loss of detail—is even more a characteristic of his designs.[2] Anyway, it may well have been partly as a result of this myopia that he pioneered the new approach to stage-designing, and persuaded his followers of the proper supremacy of colour and form over detractory details; and the sets of Reinhardt and Jacques Copeau came naturally in his wake, an influence that is of course universally apparent to-day.

So I suspect that it was no accident when Pollack (1917) found that, among 128 masters and pupils of the Fine Arts School at Paris, 48·44% were myopes and 27·34% hypermetropes, whereas in the population at large it is the hypermetropes who are about three times as numerous as the myopes, while Siegrist (1917) recorded an artist (and I have myself encountered two) who found that he could only continue good paintings by having his myopic spectacles well under-corrected.

Myopia and hypermetropia have also been said to have a direct influence on the preponderant colour that artists used. The blue rays of light are refracted more than the red, and so are brought to a focus slightly in front of the normal retina, and the red rays correspondingly just behind the normal retina; hence the myope, with his abnormally elongated eye, will see reds better, and the hypermetrope, who has the opposite deformity of a shortened eyeball, will be correspondingly better on blues, a phenomenon that has been capitalized for sight-testing in the "duochrome test". Indeed, according to Patry (1917) there is an actual shift of the spectrum in a corresponding direction; thus to the hypermetrope yellow becomes tinged with green (rather crude subjective tests do appear to confirm this). Indeed, the increasing fascination for reds in the case of Renoir (which has already been attributed to incipient cataract) might simply have been a legacy of his myopia. But it is a curious coincidence that colours from the

[1]His many self-portraits during early manhood show no glasses, but these may well have been executed within his limited focal range.

[2]A typical comment is that of *The Times* critic, who described the simplicity and severity of Craig's sets for Ibsen's "The Vikings" as . . . "harmonious in colouring, broad and massive in design". Craig himself had later recorded how tremendously a flight of steps appealed to him . . . ("when this desire came to me, I was continually designing dramas, wherein the place was architectural, and lent itself to my desire"). Mrs. Leeper also describes how . . . "in the early days, when Craig was young and unknown, among the few who understood his aim had been the poet W. B. Yeats . . . who would discourse on the poetic drama in his vivid magnetic way, peering with myopic eyes into the darkness while Craig was happy to sit and listen to him". It is tempting to suggest that the vision he so readily shared with Yeats was really a sharing of their mutual dependence on the strange structural world of the myope.

red end of the spectrum should so predominate in the paintings of Chinese and Japanese who are also predominantly myopic (the Japanese have only recently adopted a specific word[1] for blue), and Patry (1917, 1918) has listed several Swiss artists (B. and B. Baud-Bouy, Hodler, Henner, Meissonier, Estoppey, and Alice Bailly) whose colours as well as designs could be readily attributed to their relative hypermetropia or myopia.

Finally, the myopia and hypermetropia of an artist will both have a direct influence on the actual "projection" of his painting, and correspondingly on the optimum distance for viewing his work. When artists seek to record on their little rectangles of canvas a relatively small view, they can safely employ a simple geometrical perspective whose laws remain approximately accurate only for such a "narrow-angled" view, although I believe some (like Canaletto) get a wider-angled effect by basing their geometrical perspective on two points, about 10 degrees apart. However, the panoramic rendering (as in Fig. 8, taken with a wide-angled lens) does have much to recommend it, and we are reminded of that eighteenth century affectation "the Claude glass"—a convex mirror[2] through which some (like the poet Gray) preferred to view their landscapes, since it "opened them out" like a Claude painting. There is no real difficulty in using some cylindrical gnomic projection (like the conventional Mercator's map of the world), to transpose our view on to a two-dimensional canvas from the surface of the imaginary sphere encircling our heads on which it is in fact disposed. However, the wider the "angle" of our view the more necessary it is to observe its rendering from the same point as the original artist who painted it (or camera that photographed it). Even small-angled paintings have their natural distance for correct viewing—corresponding to the radius of that imaginary sphere around each of our heads on to which the seen world is projected: and for every artist this radius is fairly constant—representing the average distance from his canvases at which he works best. This distance is generally greater for oil paintings than for water colours, for outdoor paintings than studio paintings, in the long-armed, and, most important, in the long-sighted or hypermetrope. (This optimum viewing-distance is, of course, unrelated to the closeness or remoteness of the subject, but only of his canvas, and in vast paintings or murals it is often very great—the artist managing to achieve this by a great mental effort, or by frequently stepping backwards, or by working from sketches.)

On this reckoning, Wilson (1958) has deduced that Vermeer was short-sighted, having a short radius, although placing his subjects at a fair distance away, while Van Gogh similarly had a short radius (and therefore, one hazards, short-sightedness) but with his subjects at an unusually close range; Van Eyck had a very small radius indeed, while Franz Hals had a larger one, and is therefore a presumptive hypermetrope. Incidentally, almost as a confirmation of this, the preponderance of myopia among miniaturists had already been noted (Cantamessa, 1938).

And, as a last after-thought on myopia, we can recall that Goethe was also very myopic, and his theory of colour vision was among the first that contained truth; yet he refused to wear his glasses in public, and always objected to others wearing them (Hardy, 1934).

Presbyopia

Presbyopia is the last of the four "refractive errors" that may need the compensating power of spectacles. Here the natural weakness of focusing that comes with middle age causes a progressive difficulty with near vision, and a spurious long-sightedness like that of the hypermetrope.

It is true that a fuzziness, or what art historians would call "breadth", is very apparent in the latest paintings of relatively long-lived artists like Rembrandt and (especially) Titian, and this may indeed be attributed in part to a presbyopia that had rendered their canvases increasingly indistinct. For instance Rembrandt's portrait of Saskia (Fig. 9) when he was aged 27, shows a delicacy of detail and refinement of feature, which is in striking contrast to his self-portrait (Fig. 10) when aged 63, some months before he died, with the face like a rough-cast in mud. While again in the early Titians like "Noli me tangere" (Fig. 11) there is plenty of careful detailing which has all gone by the time we reach his later works like "The Flaying of Marsyas" (Fig. 12). I do not, of course, imply that I believe this change was primarily due to receding near-point of clear vision, for such "open brushwork" commonly marks the conquest of paint as a material, but at least it is credible that we should to some extent give this

[1] "ao" is Japanese for green and for blue, which are generally lumped together.

[2] These convex mirrors were used in all seriousness by the Dutch naturalistic artists of the previous century, sometimes in the form of a crystal ball which is actually depicted in certain of their studios (Wilenski, 1929) and this in turn probably derives from the earlier use of mirrors (typically by Vermeer) —a method said to originate with Titian's exploitation of Venetian glass. Gerard Douw is said (Descamps, 1754) to have manipulated with his foot a screen in which was set a concave lens bearing a grid of threads to correspond with a similar grid on his canvas.

refractive failing the blame—or perhaps the credit—for the change.

How different the world of art might have been if all these famous painters had been forced to wear glasses constantly. How secretly one agrees with the Rev. Mr. Cross, Vicar of Chew Magna in Somerset, who declared "The newly invented optick glasses are immoral, since they pervert the natural sight, and make things appear in an unnatural and false light" (Hardy, 1934), or with the enigmatic epitaph on Salvino d'Armato in S. Maria Maggiore, at Florence, "... inventor of spectacles; May God forgive him his sins".

Squint

After refractive errors, we come to the question of squint; and although we would expect little direct influence of an artist's squint upon his paintings there are some points of passing interest.

The first and doubtless the greatest "squinter" ever to be portrayed was Venus herself, whose squint is frequently described by classical poets (Foster, 1952); but we have little visible evidence of this as the painted irides of those earlier statues have all weathered away, and the late Roman statues, which had their pupils "chiselled-in", all had straight eyes.

The most famous of all squinting painters was Guercino, whose name simply means "the squinter", and whose self-portrait (Fig. 13) makes no attempt to conceal that convergent and presumably poor-sighted eye; it would be wishful arguing to aver that there was any consequent failure of the third dimension in his many familiar paintings, any more than in those of a recent P.R.A. who had only one eye. One of our most famous English contemporaries has, in fact, a small residual squint with no binocular vision; and he volunteered that in his earlier years, when his painting was more naturalistic, he did have a constant difficulty with this third dimension.

Of more interest perhaps is Dürer who had a divergent squint.[1] He made various self-portraits; that from the Louvre (Fig. 14) shows his divergent squint, with the right eye failing to swing inwards, and he is showing rather nicely the classical compensatory head-turn to the left, in order to minimize the ocular deviation and its resultant diplopia; and in the portrait from Erlangen (Fig. 15) his right hand seems appropriately to be warding off the confusing second image seen by that divergent right eye. The eyes seem to have been straight when he was 13 (Fig. 16) but the squint was well established seven years later and even more so in the various portraits of his middle age. Sometimes in the later portraits it is the left eye that seems to be diverging, but this was probably not because he had an alternating squint but a result of the drawing being completed by an assistant rather than by the artist scanning his own features through a mirror (of course in the case of the engravings, there has necessarily been a reversal of the plates). In life, we must presume that the left eye would have been the divergent one.

Various subjects of portraits as well as the artists themselves have had their squints immortalized—such as Thomas Inghirami (Fig. 17), whom Raphael has painted with his head turned slightly to the left and his eyes well over to the right—a similar compensatory posture: in his case the squint may well be the sequel to a monocular myopia which had caused the obvious prominence of his right eye; and this recalls the even grosser case of Federigo da Montefeltro (Fig. 18) whose many paintings are all done in profile to conceal the unsightly right socket, although the bulging left eye is barely more pleasing. Sometimes, however, the divergence is the result of the artist's affectation, rather than his realism, as when it is used to express ecstasy (if one converges for near vision, it would seem natural to diverge when looking heavenward—beyond infinity). Indeed it has become such a habit in El Greco's saints that they even diverge when the eyes are down-turned as with St. Simon and St. Luke, although the latter was in fact thought to be El Greco's own self-portrait (Fig. 19). This question of divergence to portray other-worldliness is interesting, since the relative positions on the two corneas of their bright light-reflections are constantly telling us that so many painters have consciously or unconsciously counterfeited a mild divergent squint (Alaerts, 1947).

An upward deviation is often used to underline dramatically the blindness of the subject, whose eyes seem to turn desperately upwards searching for the sun, as in the familiar beggars of Brueghel[2] (Fig. 21), although sometimes it is sufficient just to exaggerate the depth of the anophthalmic socket, as in Rembrandt's Tobias (Fig. 20). In this context, too, there are always

[1]We happen to know that he purchased some glasses for 9½ farthings (Howard, 1954), but unfortunately this was not until he was well in the presbyopic age (1521), and so has little bearing on his squint except to suggest that there was probably no underlying myopia.

[2]Peter Brueghel the elder is said by Torrilhon (1958) to have been the arch-diagnostician of eye ailments, the five beggars from his "Parable of the Blind" representing from left to right: ocular pemphigus with secondary corneal opacities, photophobia possibly from an active kerato-uveitis, phthisis bulbi and corneal leucomata. In "The Wedding Banquet" he has likewise depicted a myxœdematous facies, although the more famous rendering of senile ectropion (Fig. 22), formerly attributed to Brueghel, was probably by one of his predecessors.

A B

FIG. 3.

A B

FIG. 5.

A

B

FIG. 2.

A B

FIG. 4.

See page 728 for legends.

the corpus callosum, below by
allosum, medially by the sept
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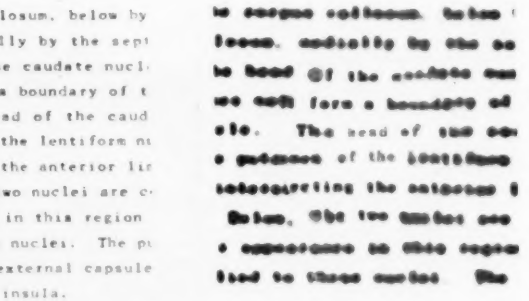


FIG. 6.

FIG. 7.

FIG. 2.—A, El Greco—"Cardinal de Guevara". B, El Greco—"Cardinal de Guevara" photographed through "astigmatic" lens at 15° (Ahlström).

FIG. 3.—A, Holbein—"Henry VIII". B, Holbein—"Henry VIII" photographed through "astigmatic" lens at 90° (Ahlström).

FIG. 4.—A, Holbein—"Dead Christ". B, Holbein—"Dead Christ" photographed through "astigmatic" lens at 90° (Ahlström).

FIG. 5.—A, Cranach—"Lucretia". B, Cranach—"Lucretia" photographed through "astigmatic" lens at 180° (Ahlström).

FIG. 6.—Relative acuity of the peripheral field. On the left is what we think we see, when looking at the centre of the page; and on the right is what we actually see (in terms of lowered visual acuity).

FIG. 7.—Matisse—"Sous Bois", 1925. (Courtesy of A. Tooth, Ltd.)

FIG. 8.—Panoramic view. ("Alfa" photograph by G. Rossat-Mignod.)

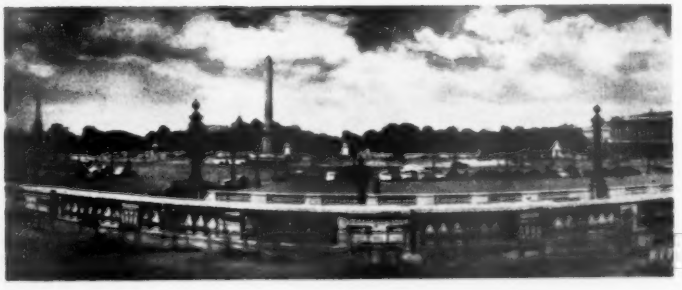


FIG. 8.

FIG. 9.—Rembrandt—"Portrait of Saskia", 1633. (Cassel Gallery.)

FIG. 11.—Titian—"Noli me tangere". (Courtesy of National Gallery, London.)

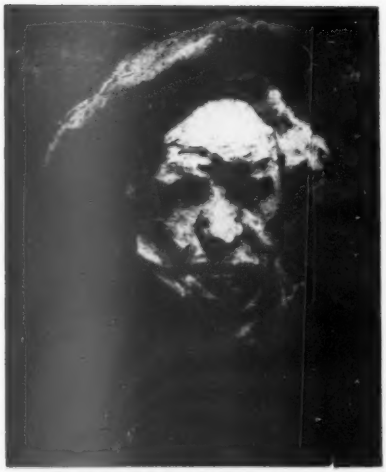


FIG. 10.—Rembrandt — Self-portrait, 1669. (Aix-en-Provence Museum.)

FIG. 12.—Titian—"The Flaying of Marsyas". (Kremsier—Archep. Gall. "Late".)

FIG. 13.

FIG. 15.

FIG. 14.

FIG. 16.

FIG. 13.—Guercino—Self-portrait. (Courtesy of D. Mahon, Esq.)

FIG. 14.—Dürer—Self-portrait, 1493. (Paris—Louvre.)

FIG. 15.—Dürer—Self-portrait, 1491. (Erlangen—Universitätsbibliothek.)

FIG. 16.—Dürer—Self-portrait, 1484, aged 13. (Vienna—Albertina.)

FIG. 17.—Raphael—"Thos. Inghirami". (Florence—Pitti.)

FIG. 18.—Piero della Francesca—"Federigo da Montefeltro". (Florence—Uffizi.)

FIG. 19.—El Greco—"St. Luke", 1604. (Toledo—Cathedral.)

FIG. 20.—Rembrandt—"Blind Tobias" (detail) 1626. (Coll. J. Goudstikker—Amsterdam.)

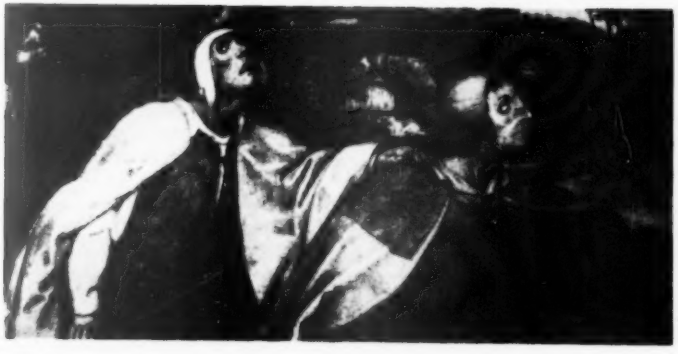
FIG. 21.—Brueghel—"The Parable of the Blind", 1568. (Naples—Mus. Naz.)

← FIG. 17.

FIG. 18.→

←FIG. 19.

FIG. 20.→



←FIG. 21.

FIG. 22.—Brueghel (?)—"Portrait of a Fool", showing senile ectropion. (Vienna).

FIG. 24.—Velasquez—"Jacob Receiving the Blood-stained Coat of Joseph", 1630. (Escorial.) Showing the effect of a "mirror-wise" reversal in the lower picture.

FIG. 23.—The Master of the Tired Eyes—"Portrait of an Unknown Woman". (Courtesy of National Gallery, Dublin.)

the artists who like to project their melancholy on to their subject by constantly featuring a droop of the upper lids (Fig. 23), like that shadowy painter who is known simply as the "Master of the Tired Eyes".[1]

Before we leave the question of squint, it is perhaps relevant to consider Huber's (1935*a*, *b*) theory of eye dominance. In western countries, where our right eye is generally the dominant one, and we read from left to right, our emphasis is apparently directed to the left side of the picture; so that there we place our principal subject, if we wish (as in most baroque paintings) to convey a feeling of tension or movement; but if the main subject is on the right, the picture becomes calmer and more static. In other words, with this painting of Velasquez (Fig. 24), the lower version, which is reversed,

[1]Probably the "Master of the Statthalterin Maria" or "Willem Scrots" or "Stretes" (Friedlander, 1935–36).

should be more restless and taut because the seated Jacob has moved across to the left. And indeed I wonder whether the opposite is true for eastern races who often write in the contrary direction, and in whom the left eye is, I understand, generally the dominant one.[1]

Colour-blindness

Major colour-blindness would hardly permit painting of any merit, but one man in every twenty is partially colour-blind, in that he has poor differentiation of red and green, so it would be surprising if we did not find some evidence of this among established painters. Just to underline this curious hereditary defect, I am reproducing (Fig. 31) the attempt of one of the senior London eye-surgeons—who happens to be red-green colour-blind—to match the upper colours of the sequence (thus in the case of 6A in Fig. 31, he could not decide which of his red and white was the better match for that bottle-green). When we look at those random matches, we can only marvel that the existence of colour-blindness was not established until 1798; and all those mediæval apprentices in mosaics and illuminating must somehow have concealed their muddles. It has, however, been noticed (Riddell, 1949) that this may well be the reason why, in recent decades, there has been such a high proportion of colour defectives in engraving (? the artist-rejects), just as there is in the Marines (? rejects from the Royal Navy).

The effect of colour-blindness on artists was first discussed when Liebreich in 1872 noted at "The London Exhibition" that year how with certain painters the roof-tops and the oxen (one suspects that 1872 was a prime year for such subject-matter) were depicted as red on the well-lit side and green on the dark side. This so-called "sign of Liebreich" of the red-green colour-blind has been emphasized by subsequent writers, notably Angelucci of Naples, who in 1908 gave an exhibition of the paintings of three such colour-defective artists. He particularly noted how on one canvas a naked child, sitting in the shadow, has come out entirely green (and the artist responsible for this admitted that red and pale green both seemed grey to him), also how the tree leaves, when lit by the sun, were not yellow-green but bright yellow, and those in the shade were blue-green or sometimes entirely blue.

Colour-blind painters, it seems, generally try and attenuate their failing by reducing their colour-content, so that their pictures often seem a little melancholy. Whistler, with his nocturnes, is an alleged example of this, and so is Carrière (Patry, 1918), with those faces of his dimly emerging from his paintings like ghosts from the darkness; Grottger, the Polish master of pencil and charcoal is a better-established instance, since he conceded that colours were right out of his reach (Majewski, 1936). On the other hand a minority of colour-defectives blithely ignore their failing, and their colours tend to be exalted (and sometimes rather irresponsible) in consequence. In 1933 Strebel claimed that at an exhibition of paintings by Léger (Fig. 33), he was so struck by the blue and yellow predominance with clay-coloured backgrounds that he diagnosed a red-green colour-blindness, and promptly tested the artist, proving himself to have been correct. The late Professor Raper was also red-green colour-blind and his water-colours, too, were all characterized by an excessive use of rather greenish blues and khaki yellows. So are two famous living painters; one of them is totally red-green colour-blind and it was once said that this is how he came to invent the blue mountains, white cottages and silver-white clouds of Irish landscapes.

Was Constable also partially red-green colour-blind? Certainly his paintings often look autumnal (Fig. 32), although he himself declared in 1833 "I never did admire the autumnal tints, even in nature . . . (but) I love the exhilarating freshness of spring"; and added that he set to portray "light, dews, breezes, bloom and freshness". If it were true, he would certainly have needed to use additional red to build up his green-matches, and it has been cogently argued (Law, 1957) that this was responsible for the brown mantle that characteristically covers his spring trees. Of course it is as easy to pick a good brown Constable tree to illustrate this, as it is to pick a good orange canvas to argue Turner's advancing cataract; but there are endless brown trees to choose from. Once Sir George Beaumont asked "Do you not consider it very difficult to determine where to place your brown tree?" and he replied "Not in the least, for I never put such a thing into a picture". I know that Sir George was referring to the brown trees which were at that period conventionally introduced, but quite manifestly Constable did introduce these, and may it not be that he did not realize how brown his trees actually were? Then in 1823 Fuseli stood before Constable's pictures in the Academy with a greatcoat on and umbrella up, to protest against their heavy overcast colouring; and ten years later he wrote to Wilkie "I like the landscape of Constable, but he always makes me call for my great coat and umbrella". Even more suggestive than the

[1]Weinstein (1958) independently observed that in far-eastern paintings, where these are bisected obliquely from bottom-left to top-right corners, the primary subject-matter is generally crowded into the upper left triangle, while the reverse is generally true of western art.

FIG. 25.—Paintings by chimpanzee (Congo), illustrating the constant general pattern peculiar to each ape. (Courtesy of Dr. D. Morris.)

FIG. 26.—Three stages in the mastery of painting by a congenitally "near-blind" boy, who had a gross peripheral field restriction (Lowenfeld, 1951).

FIG. 27.—Three stages in the mastery of sculpture by a boy who was congenitally blind (Lowenfeld, 1951).

FIG. 28.—Sculpture by a blind girl (Révész, 1950).

comments of his contemporaries are his own comments on their paintings, which he was apt to describe as insipid or vapid, since he probably saw the green as being pale or fawn-coloured (as they lacked the extra dose of red that he himself would have inserted). Others, like Farrington's landscape, he described as "heavy and crude" but added pertinently that they looked much better by twilight, for the natural colour-shift of vision in fading light would at least serve to darken the red pigment that he had used in his greens.

Constable's pictures are not, of course, all in autumnal shades; in a minority the blue-greens predominate, and white light sparkles in the foliage. Even this can be argued as confirmatory evidence if we simply attribute it to the high luminosity curve of the partial protanope (Law, 1957), and we assume that instead of the usual overcast English day, he was painting in a pale sunlight.[1]

One should, incidentally, be particularly cautious in regard to this question of Constable's colour-blindness, as I believe only one (Dedham Mill) of the large collection in the Victoria and Albert Museum has recently been cleaned, so there is bound to be some spurious darkening of the pigment due to the browning of the varnish.

Finally, here is an account I have just received from a North London art-teacher about one of her recent pupils:

"One of the students, an extremely quiet and sensitive boy, was about 17 when he joined my composition class. The first painting he produced, a street scene, showed a fair sense of arrangement, good drawing, and very unusual colour for a

[1] A further point was made by Law that many of the original sketches were in an even darker colouring than the final version; but others have doubted this with the small sketches (except perhaps the "Valley Farm"), and of the two larger sketches, comparison is difficult since the final version of the "Hay Wain" (National Gallery) has recently been cleaned.

FIG. 29

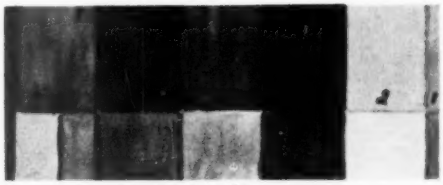


FIG. 31.

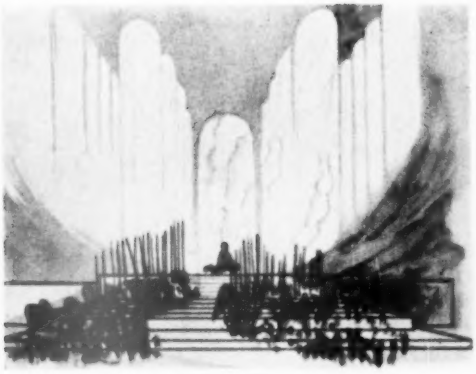


FIG. 30.

FIG. 32.

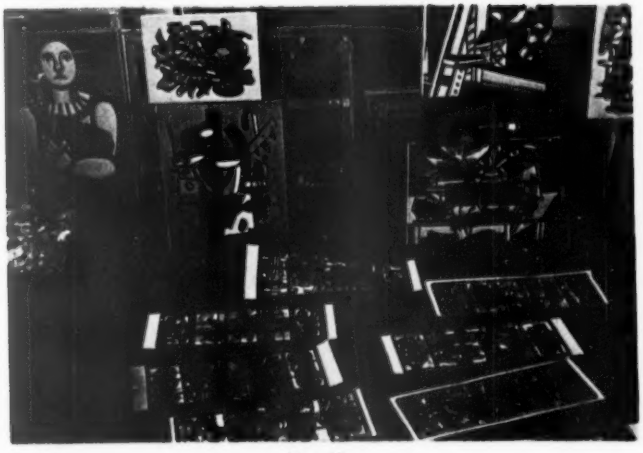


FIG. 33.

For legends see reverse

FIG. 29.—Cézanne—"Aix, Paysage Rocheux", 1887. (Courtesy of Trustees of Tate Gallery.)

FIG. 30.—Gordon Craig—"Set" for the opening scene of Ibsen's "Crown Pretenders". (Leeper; courtesy of Gordon Craig, Esq.)

FIG. 31.—Protanomalous matching. The bottom series shows the attempts of a red-green colour-blind surgeon to match the colours of the upper series.

FIG. 32.—Constable—"The Cornfield". (Courtesy of National Gallery, London.)

FIG. 33.—Léger—Designs for Church Windows at Audincourt.

FIG. 34.

FIG. 35.

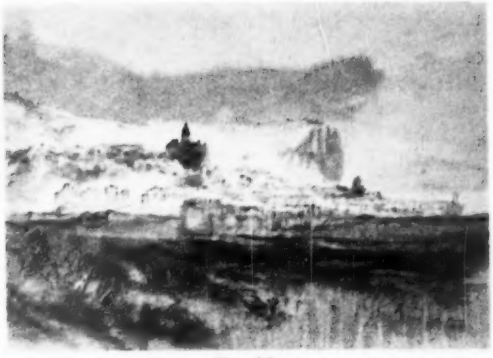


FIG. 36.

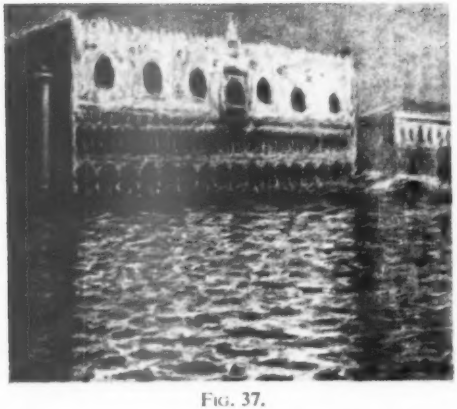


FIG. 37.

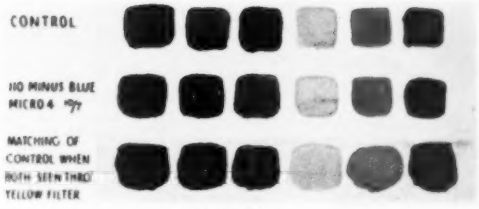


FIG. 38.

P. D. TREVOR-ROPER: *The Influence of Eye Disease on Pictorial Art.*

For legends see reverse

FIG. 34.—The effects of cataract extraction. Paintings made before and after operation. (Courtesy of Mr. F. A. Williamson-Noble.)

FIG. 35.—Turner—"On the Thames", water-colour, 1806. (Courtesy of Trustees of British Museum, London.)

FIG. 36.—Turner—"Eu", water-colour, 1845. (Courtesy of Trustees of British Museum, London.)

FIG. 37.—Monet—"The Ducal Palace, Venice", 1908. (Courtesy of Brooklyn Museum, New York.)

FIG. 38.—The effect of a brown filter on colour matching. The second row represents the artist's attempt to match the sequence of colours in the upper row when these were viewed through a brown filter. The third row represents the attempt to match the sequence of colours in the upper row when wearing such a filter as a spectacle lens, so that both upper and lower rows were viewed through it.

beginner. He used colours which were bright but not crude, with some red and yellow, a little green, and a great deal of soft blue and mauve. The general effect was exciting and pleasing; a change from the usual red, yellow, blue and meridian in their raw state used by most beginners.

"During the following lesson a week later, he asked if he could speak to me privately, and he then told me that he was completely colour-blind. He said that he knew which colours to use by the names on the tubes of paint, and that his difficulty lay in the painting of shadows. Evidently he knew, or had been told, that there was colour in shadows, but in trying to paint a cool shadow on a warm-coloured object he could not judge the quantity of the cool pigment he was adding, and often changed the original colour completely. When I saw his painting again this was obvious: a red object, for example, might be shaded in blue or purple; the colour change being so violent that the object was split completely. Although the colour values were wrong the tone values were so good that they gave the picture a unity which it would otherwise have completely lacked. All the objects in the composition, as far as I can remember, were painted in their correct colours, blue sky, red roofs, &c.

"A colleague tells me that when painting from still life the student later developed a method of working which made up for his colour-blindness. Although the objects he was painting were in front of him (in my class students worked from memory) he never actually made a mistake in identifying the colour of an object, nor did he ever ask the master to tell him. He may, however, have asked other students. His colour in this class was extremely subdued, quiet and 'safe'; there was nothing wrong with it but its extreme dullness. The form and tone were very good. He had obviously worked out a formula for colour which compensated for whatever defect he had, but which was extremely narrow and incapable of being developed. He eventually took up sculpture but I don't believe his work was outstanding."

As an after-thought on colour-vision, Cantamessa (1938) has made the engaging suggestion that the warmth of colour which different painters use depends primarily on how blond or brunette they are; since in the former, the greater amount of light that filters through the sclera gives a colder tone to the retinal image, and *vice versa*. As a crude generalization, the Nordic races do indeed seem to employ colder colour tones than the Latins, and the latter than the central Africans or Polynesians; so it would be nice to find an untutored albino to press the point. Well-pigmented races too have more yellow pigment at the macula lutea, and could therefore be argued to be relatively insensitive to the blue end of the spectrum.

And as one final after-thought, it is tempting to reflect on the apparent predilection for the red end of the spectrum in the paintings by anthropoid apes, just as there is an apparent preference for blue colours in the elaborate "paintings" of the Satin Bower-birds. It seems that in Homeric Greek, as well as recent Japanese and some primitive tribal languges to-day, there is no proper word for blue, and it has sometimes been maintained that early man had a foreshortening of the blue end of the spectrum (an engaging theory that leads straight into a lexicographic jungle which I am not competent to explore); but greens and blues are indeed strikingly absent from palæolithic cave paintings as well as from Greek and Minoan ceramics, although this is, of course, largely determined by the relative availability of the pigments they used. But the answer to this apparent preference of primal man probably lies with the psychologists, who would say that the red colours are simply the most colourful and interesting, rather than with any disorders of the "outer" eye.

The value of these sets of ape-paintings (Fig. 25) does in fact go far beyond any of the colour-discrimination which they (rather unconvincingly) displayed, for Desmond Morris has found that there is an essentially constant pattern to each individual, which can be altered with almost mathematical regularity by any pre-existing pattern on the paper; so that it may be possible to find a purely mechanistic basis for any truly abstract painting. Humans fortunately differ in having the added capacity for image crystallization and stylization, so that even the most sophisticated of our "action painters" can never aspire to this pure distillate of the simian ego.

Cataract

In most elderly people opacities form within the lens of the eye, and rarely provoke more than a little progressive blurring of vision. In addition to an overall mistiness, the advancing cataract absorbs principally the shorter spectral wave-lengths, starting with the violet and blue end of the spectrum; and ultimately it may permit little beyond the red rays to reach the retina. Conversely, after the cataract has been extracted by an operation, the sudden influx of these excluded blue rays in the presence of an established adaptation to a rosy world, may change the red vision into a temporary blue vision. This colour change is mainly apparent with the rather less frequent "nuclear" type of cataract, where the sclerosing fibres tend to become yellowish or even reddish-brown; and it is generally more striking where the patient is already myopic, so that the subsequent spectacle

lens is of comparable power to that which was needed previously.[1]

Fig. 34 is a photograph of two paintings lent to me by Mr. F. A. Williamson-Noble, of the view from the bedroom window of one of his patients; the upper one was done in August 1946; then after both his cataracts had been extracted, our artist was able, by the following August, to paint the lower version of the same view. Most arresting is the colour contrast from an overall reddish-orange to a cold greenish-blue, remembering that they were both painted in the same month of the year and the leaves did not turn early in 1946! The cataract has of course blurred the details of the upper picture, and since the right eye, with the more advanced cataract, was then virtually blind, the picture also seems flat owing to the absence of any stereoscopic vision, while there is also a diminution of size of the trees—the actual size of the retinal image being a little larger after a cataractous lens has been removed, and replaced by a convex spectacle lens in front of, instead of within, the eyeball.

With many artists a colour change towards red can be noted in their later paintings, and blithely attributed to the advance of a senile cataract. The easiest candidate for this is J. M. W. Turner, whose later pictures are well known to have become more blurred and at the same time increasingly suffused with red and orange light, so that it is difficult to avoid likening them to the upper view from that bedroom window. Fig. 35 illustrates Turner's earlier style, in striking contrast to his later impressionist phase where the details gradually disappear, and the whole paintings seem to glow with an orange-red light (Fig. 36). Since 1841 his sight was known to be failing (Monkhouse, 1899), and although I could find no specific mention of a cataract, it might well be that the Mr. Bartlett (styled "surgeon-dentist and cupper") who tended him was a little weak on ophthalmology; and at his death in 1850 we have only William Kingsley's sorry confidence to Ruskin "The simple truth is his digestion failed through loss of teeth, and he had to have recourse to stimulants, and finally took too much".

It was Liebreich (1872) who first suggested that a lens sclerosis was responsible for the changing style of Turner and of Mulready, and between the early and late paintings by this second, less interesting artist, the colour values are again noticeably different; while if we look at the earlier pictures through a yellow glass, the difference between the two almost entirely disappears, as the glass seems to correct the colour shift. And it is tantalizing to throw up a cluster of other names into whose later paintings a rosy warmth steals; Renoir perhaps, and many others.

Of these, Antonio Verrio is, I think, especially interesting. He is best known in England for his spirited mural paintings of the late seventeenth century, culminating in the famous allegory of King William III on the Great Staircase at Hampton Court. His earlier paintings are less familiar, but at the age of only 21 he had painted a ceiling in his capital town of Naples (subsequently destroyed), and in it he apparently included with macabre prescience a portrait of himself as a blind man led by a dog.

His last work in the Queen's drawing-room at Hampton Court is vividly described by Edward Croft-Murray (1959) with its rather maladroit conception of Queen Anne in glory, attended on the walls by her stumpy husband and an appropriately dormant cupid, all in a surfeit of pink colours; he concludes "... with its riot of ill-matched colours and unprepossessing faces and figures, it hardly stands as a brilliant finale to Verrio's career. Perhaps we may excuse him in part, for his sight was beginning to fail". Soon afterwards the self-portrait now in our National Portrait Gallery was completed (possibly by a friend) with its pathetic inscription "Cieco Antonio il povero Verrio"; and two years later, in 1707, he was dead. Thirteen years later the following advertisement was published by a Dr. T. Clarke, that ..."to her late majesty Queen Anne's great satisfaction Signor Verrio, the famous painter was restored to perfect sight in Hampton Court, of a blindness called gutta serena", and the evidence all suggests that this was in fact a cataract.

Another painter who frankly developed a double cataract is Monet, and the characteristic changes are very apparent in his latest paintings. Up till 1905 his whites and blues were still unalloyed, but soon after that the whites and even the greens became increasingly yellowish, and the blues more and more purple, as in "The Ducal Palace, Venice" dated 1908 (Fig. 37), while in the final pictures like those of the water-garden at Giverny, which were done after 1920 when he had turned 80, the form too becomes

[1]This experience was emphatically recorded a few months ago by a distinguished author (C. S.) when both of his cataracts were removed, and he has indeed preserved the markedly yellow cataracts that were held responsible. While an artist (H. S.) has recently written to me that, as her cataracts advanced she was increasingly puzzled by the pinks and reds in her garden, which have seemed so much brighter than usual, and how she is now impressed by the vivid crimsons and vermilions of sunset. And a patient (G. S. M.) has just volunteered that since his cataract operation "everyone appears to be wearing blue eye-shadow, their lips are purple, their hair-roots blue, and a blue haze outlines the edges of all the buildings".

vaguer as his sight has manifestly begun to fail. In 1923 he submitted to an operation that partially restored his sight, and he then started enthusiastically retouching his paintings until all his friends and relations persuaded him to desist, and in 1926 after a few days of total blindness, he died. How interesting it would be to know, as one clearly suspects, that he was "touching out" the reds and oranges that had so insidiously slipped in.

One must not, of course, forget that this colour change would only be expected in the rarer form of cataract, which becomes brownish as well as becoming opaque, and in naturalistic painters only. So, just as an example of the more usual response, I quote Sir Matthew Smith whose opaque lenses were extracted a few years ago and clear vision restored; for here there was no apparent alteration in colour values; the only changes Sir Matthew noted were that the colours had become brighter and the details clearer.

Incidentally Coverdale once submitted (1957) that age itself should normally shift our colour values towards the red end of the spectrum, as the yellow pigment at the macula accumulates, so that blue is increasingly absorbed and fails to be appreciated; and a similar spurious blue-blindness follows. As a consequence of this darkening macular pigment (which should surely have the same effect as that of the "brown" cataract) he argued that elderly painters would unconsciously have to intensify their blues, rather than relapse into the rosy vision traditionally associated with the advancing cataract.[1]

And just to balance the "cataractous" pair from the bedroom window, I have two others lent to me by Dr. J. Keevil that would seem to confirm this point. Such a change might even be said to apply to Rouault, whose obituarist commented how the " . . . claret-reds gave way to a profusion of yellow-greens". After spending his life painting "twilight", Rouault said "I ought to have the right now to paint dawn" (*Life* magazine, March 1958).

Finally, there are several people I have met, including one successful Dublin artist (H. R-C.), who see warm rosy tones with one eye, and cold bluish ones with the other, so they find themselves using one eye or the other or both according to the particular colour value they are seeking.

Retinal Hæmorrhage

In this category I would hesitantly include Sir Joshua Reynolds who went blind in one eye quite rapidly over a few weeks in the year 1789, when he was aged 65. He then hurriedly completed the female portrait he was doing, and thereafter only painted men. (Man's widespread association of blindness with sexual shame doubtless operated then as forcefully as in the more overt days of Œdipus and the Sodomites.) Even so, some months later the other eye also weakened. The doctors called it "gutta serena", which means little except that on casual inspection they could see no cataract lying white within the pupil. The probability is that he had retinal hæmorrhages which irrupted into the vitreous, since he had already suffered a paralytic stroke seven years earlier. Following the advice of his "most skillful practitioners" he abstained from pencil thereafter, lest his remaining eye should also be affected, "a determination which cost him great pain". Two years later his contemporary biographer (Northcote, 1819) describes how he "entertained strong apprehensions concerning the tumour which had been collecting for some time over his left eye" and had latterly been accompanied by much inflammation. The surgeons adopted every means (as they said) "to discuss" it, but without effect; for it was afterwards discovered to consist merely of extravasated blood, and had no connexion with the optic nerve. Then the following year he died, and as the autopsy revealed only a "præternatural enlargement of the liver", Reynolds, like Turner, had probably succumbed to an alcoholic cirrhosis.

It would have been gratifying if his later paintings had shown some characteristic form of colour change—perhaps the yellow-vision of a terminal jaundice from his cirrhotic liver if not the red-vision resulting from a cataract or from an appropriately placed retinal hæmorrhage[2]; but ever since his stroke, he had put up his prices and farmed out more and more to his pupils, giving only an occasional touch of his own brush to justify the famous signature and the famous prices. According to Waterhouse (1941) it was thought that he got Lawrence to paint the red pictures and Daniel Gardner to paint the blue ones, which leaves little scope for any scientific interpretation!

Field Defects

Into this final chapter fall those diverse diseases that silently erode the fields of vision—simple glaucoma and lesions of the various optic pathways; but it would be hard to relate such a field encroachment to any specific altera-

[1]Isakowitz (1918) had independently observed this preference for cold colours in old age and attributed it to such organic changes.

[2]The "red-vision" resulting from retinal hæmorrhage is not often noted, but one artist (T. N.) has recorded how his macula was damaged by such hæmorrhages and his paintings have all become deficient in blue (which he appreciates when this is pointed out to him).

tions in the painter's style, although various fanciful suggestions are not lacking.[1]

Simple glaucoma traditionally affects the conscientious business-man, so perhaps it is not surprising that I could find no record of any glaucomatous artist; but it is said that Greiffenhagen suffered from a central colour scotoma in his later years (attributed to his fondness for tobacco), and his later paintings bear doubtful witness to this. Then there was Wyndham Lewis who died only a few years ago with a craniopharyngioma of his pituitary that had gradually eroded his optic chiasma, and only by sitting very close was he able to complete the portrait at Magdalen College of T. S. Eliot, before the "sea-mist" (as he poignantly described it—1951) had reached inwards across his central vision, and his days of painting were done.

But where the field loss is so great that only an occasional patch of sight remains, the artist may be thrust into that haptic or kinæsthetic world which opens up a whole new system of values and a whole new trend of imagery.

For instance, these are the paintings (Fig. 26) of a little boy who only retained a tiny patch in the centre of his field of vision and so could see only an area about 2½ in. in diameter on his drawing board; he had no knowledge of the overall contours of the head that he was depicting, and in this way he was like the blind sculptor who envisages a head as the sum of a multitude of small areas which he can map out with his fingers. The particular interest of his three successive paintings is that they illustrate so clearly the three stages of development that Professor Lowenfeld (1951) described for all who are primarily subjective or autoplastic interpreters, and who are spared from having their natural haptic evaluations overlaid by the influence of good vision. The earliest stage is the diffuse representation of the whole image—apparently naturalistic because of its undifferentiated character, what Lowenfeld described as the "Stage of self-confrontation". Then after a while comes a gradual appreciation of the single elements of form and expression: "This second stage of development at some point becomes such an overwhelming discovery that it overpowers his whole concept; instead of our first, vaguely formulated, projection, we now have a structural over-emphasis of the meaningful parts. The second stage then appears of almost geometric character, since the structural element has become vitally significant in the discovery and formulation of the self." Only when he has experienced his intellectual and emotional power to express his imagery, does he move on to the third stage in which, as we see, the rigid structural and symbolic formulation gives way to a more flexible expression of visual and haptic experiences.

How nicely these compare with the stages in artistic development of the wholly blind (Fig. 27). Again the sculptural equivalent of that first crudely realistic stage is followed by the same second stage of structural discovery, with great over-emphasis of the seemingly significant features, and then comes the same final stage when the blind sculptor freely expresses his experiences by introducing new elements of form or by varying his own structural symbols.

Discussion

The diseases that have beset the great men of history have always invited speculation, especially if the greatness of their spirit was in some part coloured by the frailties of their soma; and since their exact pathology has for so long been buried with them, our guess at this, as at the song of the Sirens, generally admits a very wide solution. But we should not be deterred by this, for a spirited guess is generally beguiling and occasionally valuable. Even Berenson (1948) in his introduction to art theory suggests that "In initio erat verbum" should really be translated as "In the beginning was the *guess*".

Of the four major eye diseases that I have discussed, myopia and colour blindness at least could have an undeniable effect on the artists' rendering; the effects of cataract and astigmatism are far less certain.

The *myopic* painter, who paints without his glasses, and avoids adventitious tricks like half-shutting his eyes, using photographs or inferences, and who yet strives for a naturalistic rendering, could reasonably be expected to show just those changes of form, definition and colour that have been described. And the high frequency of myopes among artists, and among the impressionists in particular, is probably not just coincidental. Clearly not all impressionists were myopic, and there is a vast amount in impressionism apart from the myopic changes I have detailed (such as the intellectual use of complementary colours), but it remains credible

[1]Savin (1958) described how the blind half-field in a homonymous hemianopia (due to vascular lesions of the occipital lobe) may become colonized by strangers, who remorselessly gesticulate, and from whom there is no escape; or especially in arteriosclerosis of the temporal lobe, by freakish and mischievous intruders that may plague the poor arteriopath, especially if his conscience is unclear. One fanciful interpreter has attributed the malign little figures of Brueghel and Bosch to such a vascular mishap. Another whimsical interpreter has credited the brilliant auras of migraine with provoking the scintillating haloes that deck so many mediaeval saints, and even for the steps of Jacob's ladder.

that an artist's myopia may have to some limited extent thus affected his style, and set a pattern which he could himself have consciously exaggerated, or his followers could have copied.

The *colour-blind* artist again can never have a normal evaluation of the affected hues; and it seems reasonable to accept that the majority do down-tone those colours that they register less distinctly, while the minority may well, in defiance, exalt these same colours, with correspondingly less attempt at naturalism. And the evidence, such as it is, does rather support this.

In the case of the *cataractous* artist, we have obviously the same primary objection as in astigmatism, that his reddened percept should correspond equally to a normally-coloured world as to a normally-coloured canvas, so that he will surely make the canvas emerge in the same colours as the original, however he falsely imagines both to be redder than they are; so that I do not believe that astigmatism and cataract normally exert any influence, since this self-regulating effect would normally ensure that the subject and rendering did correspond. But there is just enough evidence that this does not always happen—not least those unforgettable pictures from the bedroom window—so we must briefly consider how this non-correspondence could arise.

A brown-tinted spectacle lens will cut out all the blue rays, so that blue objects then seem dirty grey, greens (which are intermediate between blue and yellow) become yellower and purples (intermediate between blue and red) become redder—as in the second row of Fig. 38; and if one tries to paint with such spectacles all the blue range of colours become less exactly differentiated and muddier (as in the third row of Fig. 38). So the artist with a brown cataract who paints rosy scenes is rather in the position of the colour-blind artist who, as I have said, generally keeps clear of the colours that seem less distinct and less colourful. On the other hand when such a cataractous painter feels compelled to use blue, he generally exalts it in order to reach through his lowered blue-perception (in this case conforming to the minority-reaction among the colour-blind); and so in this way perhaps we can explain Coverdale's apparently conflicting observation that the aged, with their heavier deposit of brown macular pigment, may accentuate their blue colours; and perhaps, too, this would account for the occasional single patch of blue that Turner interpolated among the miscellany of reds, right up to the end of his life, but always as an almost isolated hue in strong contrast to the seemingly endless variety of reds and oranges which he was using in the same picture.

But over and above this I wonder if such an artist, in spite of his distorted colour sense, may not attain a more correct valuation of the world by various secondary means. In the case of the cataractous patient, he may either have a long-established familiarity with his own pigments, and on seeing, for instance, a reddened tree, he would mix them in such proportions as he knew from the past would give that very hue, even though the canvas as a result seemed to him disproportionately red (it is said that Monet managed to compensate in part for his failing colour discrimination by having his tubes specially labelled); or else it may be the memory picture that has become established, and the patient who experiences a relative blue-vision after losing his cataract may have become so adapted to the rosy world, that he contrasts the new bluish world thus suddenly presented with that rosy memory picture of the world which had become accepted as the true coloration and over-emphasizes the blue pigment on his canvas in consequence.

However, this naturally applies only to straight descriptive painting. With original artists like Matthew Smith, who use strong colours and generalized forms, we would hardly expect any such change; for here it is a memory image, probably begotten long before the cataract started to form, which has been altered by all his complex of ideas and associations till it is registered on canvas in the form we finally see. So little wonder that here the transient colour-value changes of the outer eye are of small consequence, any more than Beethoven's deafness impeded his original compositions which derived from his long-digested imagery in sound.

When we come to *astigmatism*, and attempt to explain the distortion of shapes rather than of colour, it cannot be a question of memory, since the astigmat's distortion has normally been there from birth; and it is even more difficult to postulate any mechanism that would, in spite of our expectation, let the subject and the rendering fail to tally.

There are indeed some scraps of evidence that an astigmat does sometimes, to some extent, distort along the line of his astigmatism. The classic experiment (of Berger and Hess) can easily be confirmed, by closing one eye and wearing before the other an astigmatic lens; such an "artificial" astigmat will draw an ellipse if he attempts to draw a circle "out of his head" (that is, without having one to copy, when he will simply draw its facsimile), the circle being elongated along the line of the astigmatism; while if he tries to draw a line perpendicular to the edge of his paper, this will lean slightly in a direction which corresponds to the power and

obliquity of the astigmatic lens that he is wearing —presumably, because he is making an intellectual compensation.

But, quite apart from this I am led to wonder to what extent Lowenfeld's statement (1939) is true that one in four of us in our hearts think haptically and kinæsthetically, rather than visually, in other words making our basic assessment of objects by feel rather than by sight, just as dogs think olfactorily—in a world orientated by smells; and the oblique astigmat, whose visual world is sloping, knows from touch and intellect that it is in fact upright, and therefore paints it on his canvas, so that it seems upright—not as he actually sees it, but in the way that he knows it ought to stand; and the result of this is, of course, that the picture we see is sloping in the opposite direction. And perhaps as a confirmation that the visually handicapped do lean on their haptic sense, the few sculptures that El Greco made are more normally proportioned and show less evidence of his famous oblique distortion.

Finally, as a reminder of this haptic world—the only world to the blind—Fig. 28 shows a typical "blind" sculpture, in this case by a girl who was blinded in infancy, which illustrates well the exaggerated relief, and the long connecting links, such as the neck, which unite the various haptically significant features like the eyes and nose. If we can persuade ourselves that El Greco's distortions stem from a haptic desire to straighten his sloping percepts, might it even be that his co-mannerist Parmigiano, who always featured such long necks, had been wrought upon by the same haptic urge as the little girl in her statuary?

But I suspect that I have already strayed dangerously far from the solid shores of my own exact science into these subjective waters—so tantalizing but so treacherous—when I had only planned to splash about for a little in the shallows. At least I have given wings to the evidence, such as it is; and if there are any ultimate conclusions to be drawn—they will not be mine, but yours.

Summary.—When a naturalistic artist suffers from certain eye diseases, like myopia, presbyopia and colour-blindness, his rendering must surely be affected; other eye diseases like astigmatism, cataract, and retinal hæmorrhage might also conceivably have some influence on his style.

It is hard to marry the subjective language of the artist with the objective language of the scientist, but an attempt has been made to analyse the changes that could occur from each of these diseases, to assess the value of any interpretations in current and past ophthalmic literature, and in the light of evidence from contemporary artists who are known to be affected, and from famous masters to whom these diseases have been imputed, to try and sift that little residue of truth which still remains in all these engaging theories.

Acknowledgments.—My acknowledgments are due to many friends, ophthalmologists, artists and art historians who have helped me with ideas and evidence. Especially to Mr. Edward Croft-Murray who provoked my interest, Mrs. Frank Law, from whose extensive but yet unpublished enquiry into Constable's alleged colour-blindness I have so liberally quoted, Mr. Otto Ahlström who readily sent me all the "astigmatic" photographs taken through the anamorphic lens which his son Mr. Carl Otto Ahlström had constructed, Mr. R. C. Davenport, Mr. H. E. Hobbs, Mr. B. W. Rycroft, Dr. J. Keevil, Professor Norman Ashton, Mr. Raymond Mortimer, Mr. Carl Winter, Mr. Michael Jaffe, Mr. Jonathan Mayne, Mr. A. Wilson, Miss Winifred Taylor and many others, not excluding the various galleries and other owners of the pictures quoted or reproduced: and not least to Dr. Peter Hansell's Illustration Department at the Institute of Ophthalmology for nearly all the photographic reproductions, and to Mrs. D. Cuell for her patient secretarial assistance.

REFERENCES

AHLSTRÖM, O. (1955) *Opt. Sci. Instr. Mkr.*, **130**, 253.
ALAERTS, L. (1947) L'Oeil Humain dans l'Art. Brussels.
ANGELUCCI, A. (1908) *Rec. Ophtal.*, **30**, 1.
BERENSON, B. (1948) Æsthetics and History. New York.
BLANSHARD, F. B. (1949) Retreat from Likeness in the Theory of Painting. New York.
BOZZOLI, S. (1957) Otium (l'occhio del mio amico poeta). Treviso.
CANTAMESSA, G. (1938) *Boll. Oculist.*, **17**, 1035.
COVERDALE, H. (1957) Personal communication.
CROFT-MURRAY, E. (1959) Decorative Painting in England, 1537–1837. London.
DESCAMPS, J. B. (1754) La Vie des Peintres flamands, allemands et hollandais. Paris.
EHRENZWEIG, A. (1953) The Psycho-analysis of Artistic Vision and Hearing. London.
FISCHER, E. G. (1783) Quoted by Ahlström, O. (1955).
FOSTER, J. (1952) *Trans. ophthal. Soc. Austr.*, **12**, 28.
FRIEDLANDER, M. J. (1935–36) Die Altniederlandische Malerei. Berlin: **12**, 24; **13**, 90.
GREEFF, R. (1915) *Dtsch. opt. Wschr.*, **124**.
HARDY, W. E. (1934) Random Reflections. London.
HOWARD, J. R. (1954) *Guildcraft* (June), 21.
HUBER, O. (1932) *Klin. Mbl. Augenheilk.*, **89**, 97.
—— (1935*a*) *Klin. Mbl. Augenheilk.*, **95**, 574.
—— (1935*b*) *Z. Augenheilk.*, **86**, 404.
ISAKOWITZ, J. (1918) *Klin. Mbl. Augenheilk.*, **61**, 454.
—— (1933) *Klin. Mbl. Augenheilk.*, **91**, 110.
LAW, B. M. (1957) Constable and Colour. (Paper read during the Canadian Ophthal. Soc. Congr. at Banff, Alberta.)
LE GRAND, Y. (1952) Optique Physiologie. Paris.
LEEPER J. (1948) Edward Gordon Craig. Harmondsworth.
LEVI-SANDER, E. (1917) *Klin. Mbl. Augenheilk.*, **59**, 657.
LEWIS, W. (1951) *The Listener*, May 10.
LIEBREICH, R. (1872) *Not. Proc. roy. Instn.*, **6**, 450.
LOWENFELD, V. (1939) The Nature of Creative Activity. London.
—— (1951) *J. Æsthetics and Art Criticism*, **10**, 1.
MAJEWSKI, K. A. (1936) *Ann. Oculist., Paris*, **173**, 554.
MARQUEZ, M. (1926) *Arch. Oftal. hisp.-amer.*, **26**, 715.
—— (1929) *Arch. Oftal. hisp.-amer.*, **30**, 249.
MILLS, L. (1936) *Arch. Ophthal., Chicago*, **16**, 208.
MONKHOUSE, C. (1899) Dictionary of National Biography. London: **57**, 348.
NORTHCOTE, J. (1819) Life of Sir Joshua Reynolds. 2nd ed. London.
PATRY, A. (1917) *Klin. Mbl. Augenheilk.*, **58**, 597.
—— (1918) *Ann. Oculist., Paris*, **155**, 60.
POLLACK (1917) Quoted by Patry (1917).
RÉVÉSZ, G. (1950) Psychology and Art of the Blind. New York.
RIDDELL, W. J. B. (1949) *Proc. R. soc. Med.*, **42**, 145.
SAVIN, L. (1958) *Trans. ophthal. Soc. U.K.*, **78**, 315.
SIEGRIST, A. (1917) *Klin. Mbl. Augenheilk.*, **58**, 601.
STREBEL, J. (1933) *Klin. Mbl. Augenheilk.*, **91**, 258.
TORRILHON, T. M. (1958) M.D. Thesis, Paris (Quoted in *Time* magazine, 12.2.58).
VILLA ORTIZ, J. M., Jr. (1934) *Rev. Ophthal. S. Paulo*, **3**, 203.
VOLLARD, A. (1925) An Intimate Record. Trans. by H. Van Doren and R. T. Weaver. New York.
WATERHOUSE, E. K. (1941) Reynolds. London.
WEINSTEIN, P. (1958) Szemünk Világa. Budapest.
WILENSKI, R. (1929) Dutch Art. London.
WILSON, A. (1958) A New Theory of Perspective. (Yet unpublished monograph.)

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Meeting
February 23, 1959

Giant Cell Reparative Granuloma

By H. P. Cook, M.B., F.D.S. R.C.S., M.D.S.
London

Giant cell lesions of the jaws still present a difficult problem in diagnosis for the clinician. The histological appearance of these lesions taken by itself may be unhelpful so far as reaching a definite conclusion is concerned. There are several conditions, for example, cherubism, hyperparathyroidism, renal rickets and osteoclastoma, in which multinucleated giant cells are found in a fibrous tissue stroma. The final diagnosis therefore usually depends upon such additional factors as the site of the lesion, the age of the patient and the level of certain serum values considered in conjunction with the histology.

In recent years the condition of giant cell reparative granuloma of the jaws has been recognized and defined as a separate entity from the true osteoclastoma. Jaffe (1953) drew attention to the condition, and distinguished it from osteoclastoma on both clinical and histological grounds. He considered that clinically reparative granuloma occurred more commonly in individuals under 25 years of age, that it was not aggressive, in the sense of possible recurrence or metastases, and that it responded well to simple curettage or radiotherapy. Histologically the giant cells were small and sparse, tending to be focally arranged around areas of hæmorrhage. Delicate trabeculæ of new bone or osteoid might also be present in the spindle cell stroma. He related the giant cells present to areas of hæmorrhage rather than regarding them as tumour elements. He admitted, however, that it was difficult to reconstruct completely the stages of a reparative process.

Two cases of the condition, one in the maxilla and one in the mandible, were reported by Umiker and Gerry (1954). Both of these were treated by curettage and in each case about two pints of blood was lost, a point which emphasizes the vascularity of the lesions. These authors agreed with Jaffe's views and published a table comparing the characteristics of reparative granuloma and osteoclastoma.

A further case, affecting the mandible, was reported by Newton (1957) and Radcliffe and Friedmann (1957) presented 2 cases, both of the maxilla, which were successfully treated by surgery. It is difficult, however, to accept the view put forward in the latter paper that the condition "affects almost exclusively the maxilla", as no large series has yet been published. The opinion is difficult to substantiate even if one considers those series published as giant cell tumours of the jaws. For example, Berger (1947) reported such a series in which 4 cases might be acceptable as reparative granulomata on grounds of age group, clinical behaviour and histology. 3 of these involved the mandible and one the maxilla.

Case Report

A schoolboy aged 11 years who was symptomless was referred by his dental surgeon who had noticed an abnormal shadow in the left mandible on a routine X-ray.

On examination.—There was a swelling of the left lower alveolus which held the first and second incisors, a premolar and the first molar. The swelling extended from the second incisor to the first molar and was about 1½ in. (4 cm.) long. The bone was expanded mainly in a buccal direction and this produced a hard smooth swelling with a suggestion of surface lobulation. The covering mucosa had a bluish appearance at one point, but was otherwise normal. The swelling was painless and, on firm pressure, elastic.

External palpation did not suggest involvement of the mandibular body. No lip numbness was present and no significant lymph nodes were felt.

X-ray investigation revealed a multicystic area extending from second incisor to first molar. The outlines of these cavities were well defined and evidently represented bony septa. The lesion involved the whole width of the mandible extending from alveolus to lower border (Fig. 1). Occlusal view showed considerable buccal deposition of new bone and a concentric linear pattern suggesting successional periosteal activity (Fig. 2). X-rays of skull, pelvis, chest, hands and long bones were normal. A drill biopsy was next carried out under local anæsthesia. This showed fragments of a spindle cell matrix throughout which were numerous multinucleated giant cells and macrophages containing hæmosiderin. Further investigations were carried out in hospital: Repeated serum values for calcium, phosphorus, alkaline phosphatase, urea, and proteins were within normal limits. A calcium balance test was performed and found normal. The child's

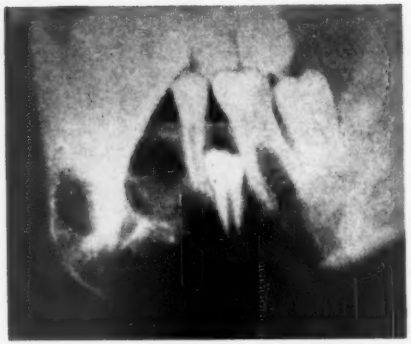

FIG. 1.—Radiograph of left mandible—cystic areas extending through whole depth of mandible.

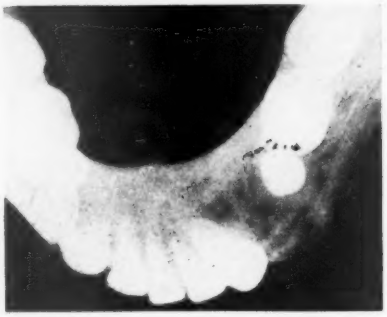

FIG. 2.—Radiograph of mandible, occlusal view—widening of alveolus and body, displaced teeth, periosteal activity.

parents were also X-rayed, and films of their jaws were normal. It was decided that this was a case of giant cell reparative granuloma and that treatment should consist of curettage. This was performed on June 6, 1958.

A large intra-oral flap, including mucosa and periosteum, was reflected from incisor to molar region and |34567 were extracted. Bone was removed to expose the soft friable tissue of the lesion. This was gradually curetted out leaving bony loculi with smooth walls. These were joined together by removing the septa, confirming eventually that the disease extended almost to the lower border of the mandible.

The inferior dental and mental nerves were identified and retracted out of danger. The buccal plate and spongiosa were then removed from the incisor to the molar region and to within about $\frac{1}{2}$ in. (1·25 cm.) of the lower border. Hæmorrhage was considerable but was controlled by hot saline packs. 25 oz. of blood was lost and 1 pint of blood was given intravenously immediately after operation.

Finally the bony cavity was packed with bone chips made from "quick freeze" homogenous stored bone. The flap was replaced and completely sewn up with interrupted sutures.

Histology of specimen.—This was essentially similar

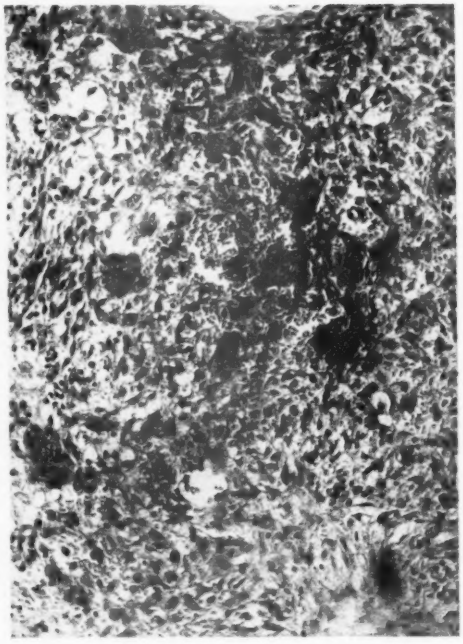

FIG. 3.—Specimen. H. and E. × 125. Giant cells showing focal distribution.

to that of the drill biopsy. The giant cells were focal in distribution and the stromal cells had moderately abundant cytoplasm. Occasional areas of hæmosiderin were present (Fig. 3).

Progress.—A seven-day course of crystalline penicillin was given, 500,000 units i.m. b.d. There was remarkable absence of soft tissue swelling following the operative trauma. Primary union of the incision took place and sutures were removed on the seventh post-operative day. Hæmoglobin was 76% and a course of oral iron was given. Total admission

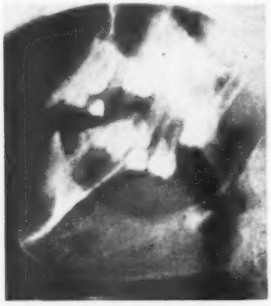

FIG. 4.—Radiograph of mandible, eight months post-operative, showing consolidation of graft.

period was twenty days. After a period of eight months the graft is well consolidated with new bone trabeculæ present in the operation area. The alveolus has remodelled well. Lip sensation is normal. So far there is no recurrence of disease (Fig. 4).

Conclusions.—The case illustrates well the vascularity of the condition confirming the experience of other authors. It also demonstrates that radical resection is unnecessary for giant cell reparative granuloma, at any rate in the first instance.

The response to treatment shows that "quick freeze" homogenous stored bone chips can be successfully used by an intra-oral approach to repair bony defects. It seems reasonable to explore the method further in suitable cases, without resorting to long preparatory courses of antibiotics as recommended by Cohen (1955) and Green (1958).

Acknowledgments.—I should like to thank Mr. R. Sutton Taylor for his kindness in allowing me to treat this case and make this report. I am also grateful to Dr. A. Morgan for the photomicrograph and Dr. P. Kerley for the X-rays.

REFERENCES

BERGER, A. (1947) *J. oral Surg.*, **5**, 154.
COHEN, B. M. (1955) *J. oral Surg.*, **13**, 34.
GREEN, H. G. (1958) *Oral Surg.*, **11**, 453.
JAFFE, H. L. (1953) *Oral Surg.*, **6**, 159.
NEWTON, E. A. (1957) *Oral Surg.*, **10**, 11.
RADCLIFFE, A., and FRIEDMANN, I. (1957) *Brit. J. Surg.*, **45**, 50.
UMIKER, W., and GERRY, R. G. (1954) *Oral Surg.*, **7**, 113.

A Dental Appliance for the Production of Artificial Voice

By R. V. TAIT, B.Sc., L.D.S. R.C.S.
Rickmansworth

THIS appliance has been invented in an attempt to find a satisfactory method whereby patients may produce intelligible speech by artificial means. It is of value to those patients who have lost the use of the vocal cords. The majority of such cases are those where the larynx has been removed in the operations of laryngectomy or laryngopharyngectomy, but the appliance may also find applications in conditions where the vocal cords, though present, cannot function; e.g. in cases of paralysis of the cords, or obstruction of the larynx or trachea relieved by permanent tracheotomy.

Complete loss of voice is a major handicap comparable with blindness and deafness in the sense of isolation it may produce in some patients. Fortunately, the majority of patients who undergo laryngectomy learn to speak by "œsophageal voice". Air is taken into the pharynx or œsophagus and then audibly regurgitated into the mouth. Intelligible speech can be produced by this means. However, those who undergo laryngopharyngectomy, with loss of musculature at the lower part of the pharynx, find the production of œsophageal voice more difficult. This appliance is intended for those who fail to learn the œsophageal method of speaking.

The six most important features of any satisfactory artificial aid to speech are as follows: (1) The appliance must set the air in the mouth in vibration at audible frequency and volume, the fundamental frequency falling within the normal voice range of the patient with overtones resembling as closely as possible those of natural voice. In brief, the artificial voice should sound like natural voice. (2) The appliance must combine clear intelligibility of speech with simplicity of operation. (3) It must be small and easily portable, inconspicuous and generally convenient to use. (4) It must be thoroughly reliable in use, and require little in the way of servicing to maintain it in satisfactory operation. (5) It must be reasonably priced. (6) It must be reasonably economical to operate, not requiring very frequent replacement of expensive batteries.

The new appliance.—Until some better name is suggested, I am referring to the new appliance as an oral vibrator, for the following reasons. There are a number of artificial speech aids which depend for their operation on a vibrating diaphragm external to the mouth. The sound from the diaphragm is usually conveyed into the mouth either by means of a tube or by conduction through the tissues. In this country, these gadgets are generally referred to as "vibrators". I have named my new appliance the oral vibrator because it also produces its sound by means of a vibrating diaphragm, but in this case the diaphragm is worn inside the oral cavity.

The appliance consists of an artificial palate into which is sealed an electromagnetically vibrated diaphragm. In patients who normally wear an upper denture, the appliance is conveniently incorporated into the palate of the upper plate. The coil of the electro-magnet is connected by a fine twin flex coming out of the corner of the mouth to a small box containing a transistorized audio-oscillator (Fig. 1). When the switch on the box is depressed the diaphragm

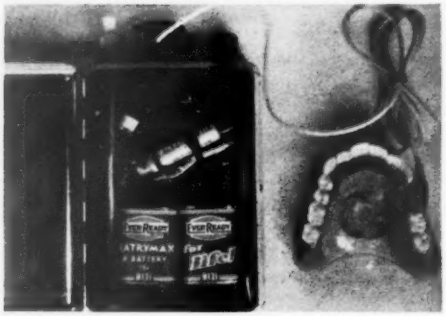

FIG. 1.

in the palate is set in strong vibration, producing an audible note. All that is necessary to produce speech is for the patient to perform normal speech movements of the tongue, lips and jaws, and the sound produced by the diaphragm becomes modulated into words.

I will now consider the extent to which this appliance possesses the six features I have suggested as desirable in an artificial speech aid.

(1) *Naturalness of voice.*—The appliance does not closely simulate natural voice, having a mechanical "buzzer-like" quality. None the less, the quality of voice produced by this appliance compares favourably with that of any other artificial voice I have yet heard. It is very simple to construct the oscillator in such a way that the pitch of voice can be varied by turning a knob on the box, allowing the normal inflections of speech to be closely copied, thereby increasing both the intelligibility and naturalness of voice. Experience so far has shown, however, that patients are not interested in this refinement, preferring to operate the appliance in the simplest possible way even though this produces a less natural quality of voice.

(2) *Intelligibility combined with simplicity of operation.*—Completely intelligible speech is possible with very little practice. Clarity is increased by interrupting the sound between words and for the unvoiced consonants, and the assistance of a speech therapist is valuable in giving the patient a little instruction on these lines. However, the intelligent patient quickly learns to synchronize the operation of the switch with the words, and this appliance is probably the simplest in operation that has yet been devised.

(3) *Convenience.*—The only part of the appliance that need be visible is the flex connecting the electromagnet with the oscillator, and it is therefore less conspicuous than any of the external vibrators. The flex can be made easily detachable from the palate and need only be connected when the appliance is actually being used for talking. The oscillator is comparable in size to a normal hearing-aid amplifier and is conveniently carried in a pocket. One hand is kept in the pocket while talking to operate the switch.

It has not been found that there is any danger of biting the flex, which is attached to the appliance in the buccal sulcus and does not pass between the teeth. But I should point out that I do not advise eating with the appliance in the mouth. The diaphragm is sealed into the palate by a sheet of thin rubber, and if the sealing is damaged the appliance will cease to operate satisfactorily.

(4) *Reliability.*—So far, the only part of the appliance that has given any trouble has been the flex, the weak part of so many electrical appliances. With prolonged use, the flex may break at its attachment to either the palate or the oscillator. However, as the flex is easily detachable at either end, the patient is provided with a spare flex with plugs attached so that an immediate replacement can be made when necessary.

No fault has yet developed in any other part of the appliance. There is no doubt that in course of time the sealing of the diaphragm will deteriorate and require replacement, but this is quite simple to do.

(5) *Cost.*—As the intra-oral part of the appliance must be individually made to fit each patient, requiring the services of a dental surgeon, it cannot be very cheaply produced. It does not, however, contain any very expensive components and the chief factor in determining the cost is the time and skill required for its construction. The cost of the appliance is not out of proportion to its usefulness, and is comparable to that of a hearing-aid.

(6) *Running costs.*—These depend on the type of batteries used. A 9- to 15-volt supply is required and there is no commercially-produced battery that is really suitable. The appliances I have supplied to patients have made use of two Ever-Ready B.121 batteries in parallel and the appliance works very satisfactorily on these. They provide 15 volts and are very small, making it possible to keep the size of the oscillator to a minimum. However, they are certainly not designed to give the fairly large current of approximately 150 milliamps required by this appliance, so their life is short and running costs correspondingly high. A very talkative patient making continuous use of the appliance found that battery replacements cost him about 10s. per week.

If larger cells are used, the running costs can be very greatly reduced, and a 12-volt battery made up of cells of the D.14 type would be far more economical, while still being sufficiently small to

fit into a conveniently sized oscillator. Unfortunately, manufactured batteries of this capacity are not a convenient shape for a small pocket oscillator, being square rather than the flat shape which easily goes into the pocket. If one is prepared to make up batteries of suitable shape from eight D.14 cells, the running costs become trifling.

In discussing these six points, I have tried to make a fair evaluation of the appliance, mentioning both its merits and its shortcomings. In a very limited field it can be of great value to patients suffering from a distressing affliction, greatly increasing the possibility of normal life. Full details of the method of construction of the appliance have been published in the *British Dental Journal*.

In conclusion, while pointing out that this is a thoroughly practical appliance which enables a patient to talk fluently with very little training, I would suggest that there are two main lines for further improvement. First, the quality of the artificial voice needs to resemble natural voice more closely. There are several methods whereby sound can be produced inside the mouth, and experiment with these, with various designs of diaphragm and with different methods of constructing the intra-oral component, may lead to improvement in tone. Secondly, a great advance will be made if the flex can be dispensed with. This is already within view. I have constructed an experimental prototype which operates by radio-control, no wire connexion to the intra-oral component being necessary. This, of course, requires extreme miniaturization of components and batteries. Although I have contrived to get the very bulky prototype into my own capacious mouth, this model has certainly not yet reached a stage at which it is suitable for use by a patient. I hope, however, to be able to report further progress on these lines as suitable miniature components become available.

REFERENCE

TAIT, R. V. (1959) *Brit. dent. J.*, **106**, 336.

Four Cases of External Facial Sinuses of Dental Origin

By DONALD WINSTOCK, F.D.S., B.D.S., M.B.

London

DISCHARGING facial sinuses of dental origin are relatively uncommon. The subject has been reviewed by Anderson (1937), who quoted 2 cases of his own, one with a mental, the other with a cheek sinus. Stones (1954), Bailey and Love (1959) and Thoma (1958) illustrate cases with mental sinuses, whilst Pangman and Gurdin (1953) reported 3 young females with cheek sinuses. The median mental sinus associated with a lower incisal apical abscess closely simulates a sinus of fissural or infected sebaceous cystic origin and is, according to Ormsby and Montgomery (1954), the commonest variety of dento-facial sinus. Sinuses on the cheek have been mistakenly diagnosed as epidermoid cysts, actinomycosis, infected acne and even carcinoma.

The anatomic basis for these sinuses has been investigated by Endleman (1927), who believes that both gravity and the relationship of the infection to the deep cervical fascia determine the eventual site of discharge. Stones relates the site of discharge to the attachments of the buccinator muscle, perforation of the alveolar bone above the upper or below the lower attachments being conducive to fistula formation.

Case I.—A 15-year-old female presented with a discharging sinus on the right cheek. Four months previously, the patient noticed a bluish area on this cheek, preceded by mild, transient toothache. A discharging sinus soon developed, for which local treatment proved unsuccessful and she was referred to the skin clinic and from there to the dental department. Clinically, the lesion consisted of a reddish-purple papilla (Fig. 1) which contained a central punctum from which pus could be expressed by gentle pressure. The surrounding skin was dimpled and partially adherent to the deep tissues. The mouth contained numerous heavily filled teeth, but no bony swelling or soft-tissue lesion, although a firm cord was palpable from the upper right premolar sulcus to the sinus.

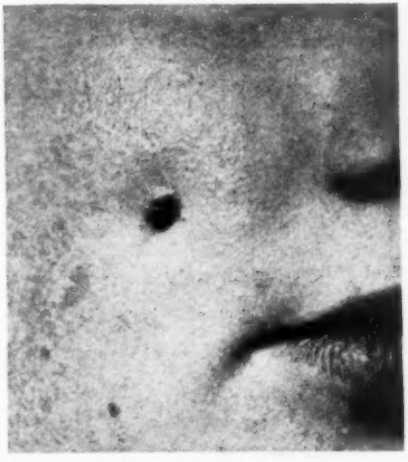

FIG. 1 (*Case I*).

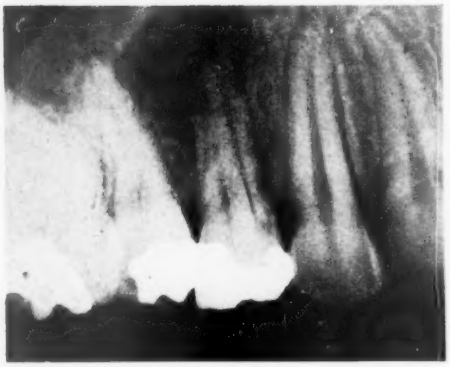

FIG. 2 (*Case I*).

X-rays disclosed an area of rarefaction around the apices of the upper right first premolar not involving the antrum (Fig. 2). At operation, under general anæsthesia, the premolar was removed and a blunt silver probe passed along the facial sinus was seen to enter the apex of the socket. The facial lesion was excised and the sinus tract curetted, the wound being sutured without drainage. Penicillin was administered for five days. Healing in the mouth and on the face was uneventful and two months post-operatively only a faint scar remained on the face. Histological examination showed the sinus to be lined by non-specific chronic inflammatory granulation tissue.

Case II.—A male, aged 23, presented with a three-months history of a discharging sinus on the right cheek, preceded by intermittent toothache in the lower right jaw lasting three months, for which treatment was not sought. Local treatment produced transient drying-up of the sinus but the patient was eventually referred to hospital because of recurrence. A papilla was present on the right cheek at the lower border, and a punctum discharged copious yellow pus (Fig. 3). A grossly carious lower right first molar was seen, and X-rays confirmed the presence of apical infection on both roots. A parulis discharged on the labial gingiva adjacent to this tooth, and a cord was palpable between the sulcus and facial sinus. The molar was removed and continuity between the sinus and socket confirmed by probing. Within ten days of extraction the sinuses had dried up, but the dimpled recess on the skin remained.

Case III.—A male, aged 22, developed a discharging swelling on the left cheek four weeks after the removal of the upper left second premolar. This lesion, diagnosed as a cyst (Fig. 4), was excised on three occasions during the next two years, but the discharge continued for another two years after which an acute flare-up occurred. X-rays disclosed a retained abscessed upper left second premolar. Removal of this root resulted in a cessation of discharge and two months later the lesion was excised. Histological examination revealed a chronic inflammatory sinus.

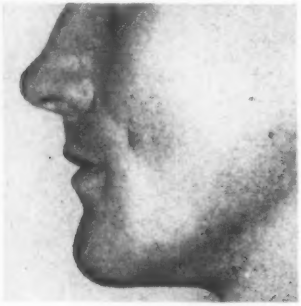

FIG. 4 (*Case III*).

Case IV.—A female, aged 24, had a lesion on the right cheek of five months' duration, diagnosed as a sebaceous cyst (Fig. 5). Excision was followed by recurrence of discharge. At a second operation, the sinus was probed and found to lead to the alveolar bone. X-rays confirmed apical infection involving both upper right premolar teeth, which were subsequently removed three days after sinus excision, no further recurrence ensuing.

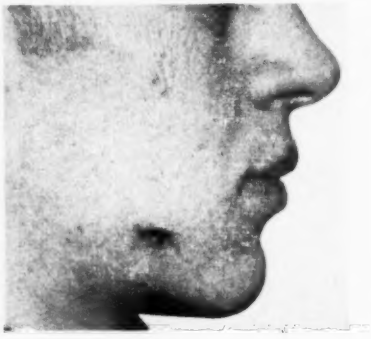

FIG. 3 (*Case II*).

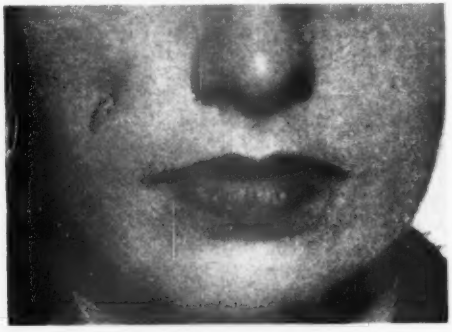

FIG. 5 (*Case IV*).

Discussion.—All 4 cases presented involved the 15 to 25 age group and all the sinuses occurred on the cheek. Case II is interesting in that sinuses appeared on both cheek and gingiva, possibly due to independent discharge of the two radicular abscesses. From a diagnostic standpoint, it would seem that facial sinuses of dental origin are often mistakenly diagnosed in the early stages, an infected cyst being the commonest initial diagnosis. Anderson stresses the paucity of information in dermatological literature on the dental aspects of facial sinuses and this is undoubtedly one of the main reasons for the frequent delay in diagnosis.

It is significant that none of these patients presented as a dental case initially.

Treatment can be either minimal, by the removal of the offending tooth, or radical, by additional excision of the sinus and its tract. Case II illustrates that removal of the tooth alone may cause regression of the sinus, but the fibrosis will result in a dimpled scar, if left untreated. Thus, it is suggested that all such scars should be excised since they have a poorer æsthetic quality than a planned linear surgical scar. Experience indicates that tooth extraction and sinus excision may be performed concomitantly, provided that an antibiotic is administered.

Summary.—4 cases are presented to illustrate the spread of infection from dental abscesses to the skin and they serve to emphasize the importance of early recognition of the possible association between face lesions and underlying dental pathology. It is advisable to examine the teeth in all cases of facial discharging lesions which persist despite simple measures of treatment as for any non-specific dermal abscess. Treatment by removal of the infected tooth, though often eliminating the discharge, will not always prevent disfiguring scar formation and surgical excision of the sinus is therefore indicated for æsthetic reasons.

Acknowledgments.—I wish to thank Dr. H. Wallace for referring Case I and Mr. Richard Battle for making available to me the case histories of Cases III and IV. Mr. John Hovell extended to me helpful advice on the preparation of this paper and the Radiological and Photographic Departments of St. Thomas's Hospital kindly provided the material for illustration and Dr. J. Burston the histological report on Case I.

REFERENCES

ANDERSON, N. P. (1937) *Arch. Derm. Syph., Chicago*, **35**, 1062.

BAILEY, H., and LOVE, McN. (1959) A Short Practice of Surgery. 11th ed. London; p. 108.

ENDLEMAN, J. (1927) Dental Pathology. 2nd ed. St. Louis; p. 264.

ORMSBY, O. S., and MONTGOMERY, H. (1954) Diseases of the Skin. 8th ed. London; p. 524.

PANGMAN, W. J., and GURDIN, M. (1953) *Plast. reconstr. Surg.*, **11**, 444.

STONES, H. H. (1954) Oral and Dental Diseases. London and Edinburgh; p. 402.

THOMA, K. (1958) Oral Surgery. London; p. 807.

Retinal Anlage Tumour [*Summary*]

By T. CRADOCK HENRY, F.D.S., M.R.C.S., L.R.C.P., and MARTIN BODIAN, M.D., M.R.C.P.

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MR. T. CRADOCK HENRY and Dr. Martin Bodian showed a case of retinal anlage tumour occurring in a male infant aged 7 months who was treated in The Hospital for Sick Children, Great Ormond Street.

Clinically, the case responded to simple excision of the tumour with no recurrence after two years.

Dr. Bodian, in discussing the pathology, said he had made use of special staining techniques in an effort to identify neurological or astroglial elements in the tumour, and also compared these sections with several controls including normal brain.

While the controls produced good staining reactions with the appropriate stains, no such reactions were obtained with the sections of the tumour. It was therefore concluded that the hypothesis of the origin of this tumour from ectopic retinal tissue could not be supported, and that this tumour should be regarded as a benign lesion in the nature of an odontomatous hamartoma.

[This paper will be published in full in the *British Journal of Surgery*.]

Meeting
March 23, 1959

Isotope Studies on the Permeability of the Dental Enamel to Small Particles and Ions

By J. L. HARDWICK, M.D.S., Ph.D., F.D.S., and J. H. FREMLIN, M.A., D.Sc.
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SINCE the beginning of this century a revolution has occurred in our conception of the structure and physiology of dental enamel. At that time, it was generally accepted that the formed enamel of an erupted tooth was a static tissue with no organic content. Even now, we cannot regard the enamel as a vital tissue if we assess vitality by the usual gross criteria applied to cells such as growth, metabolism, irritability, repair and reproduction. It is, however, undoubted that it is a dynamic tissue in which changes in its constituents can and do occur after eruption. The facts, as we know them, suggest that these changes are caused mainly by relatively simple physico-chemical mechanisms, such as diffusion or osmosis, which are intimately related to the tissue's permeability. It has, however, been claimed that enzymatic reactions occur within the enamel or on its surface (e.g. Aitken, 1949).

The permeability of the enamel has been intensively studied during the last twenty-five years by methods based on diffusion, osmosis and electrophoresis, combined in most cases with the use of distinguishable particles, such as dyes, easily recognisable chemicals (e.g. silver nitrate), toxins and radioactive tracers. Many of these experiments, both *in vivo* and *in vitro*, have shown that certain particles can pass through the full thickness of the enamel but most of them were not suitable for demonstrating that the passage occurred through intact mature enamel in contradistinction to that through damaged enamel, through cracks in the tissue or along the lamellæ. For this purpose the examination of histological sections, combined with the use of particles in the form of intensely staining dyes, easily distinguishable chemicals such as silver nitrate, or radioactive tracers and autoradiographs, is necessary. Unfortunately, most dyes have large molecules: often they have been shown to enter areas of intact, fully mature enamel around the enamel spindles and tufts and occasionally they penetrate other areas. They rarely, if ever, pass through the full thickness of intact mature enamel except along the lamellæ or cracks. Radioactive tracers are especially suitable for this type of research because many particles from small ions to large molecules can be labelled; such methods have shown that the enamel is completely permeable to certain small ions.

It has been stated (e.g. Atkinson, 1947) that the direction of the "currents" in the enamel will be influenced by the relative osmotic and hydrostatic pressures of the tissue fluids in the dentine and of the environment on the external surface of the enamel and that these "currents" may carry particles, to which the enamel is permeable, into or through the enamel. The osmotic pressure of the tissue fluids in the dentine is said to be slightly greater than that of the saliva and it has, therefore, been deduced that osmotic "currents" will occur from the saliva inwards towards the dentine. On the other hand, when highly concentrated sugar collects on the enamel surface, very high osmotic pressures develop. It has similarly been deduced that such osmotic pressures will produce strong osmotic "currents" from the dentine towards the external surface. Assumptions regarding the direction of the currents in the enamel are not justified until the level of permeability of this tissue is known. Thus if the enamel were permeable to glucose, glucose would diffuse into the enamel, together with many other small ions and particles, unaffected by the weak osmotic gradients produced by the enamel being impermeable to other particles. A knowledge of the particles which are able to penetrate enamel is therefore essential to the understanding of its physiology.

Knowledge of the permeability of enamel to glucose molecules and fluoride ions is especially desirable: glucose molecules, because their size appears to be about the limiting size of permeable particles and because they provide a common source of energy for the bacteria of caries and may be broken down by them to acid end-products: fluoride ions to account for the observations that the average fluoride content of enamel of erupted teeth is higher than that of unerupted teeth (Jenkins and Speirs, 1954; Isaac *et al.*, 1958).

Permeability to Glucose

Preliminary *in vitro* studies by autoradiographic methods of the permeability of the enamel to ^{14}C-labelled glucose (in which bacterial metabolism was reduced by raising the osmotic pressure with 20% neutral glucose, by carrying out the investigations at 3° C. and by the addition of soluble penicillin to the glucose) suggest that

glucose, or breakdown products of glucose formed under such conditions, penetrates rapidly throughout the full extent of any carious lesion which can be recognized clinically or by routine optical microscopical methods. In several teeth, in which no clinically recognizable carious lesion was present, radioactivity was observed within the pulps but this penetration may have occurred through localized areas of defective enamel or along lamellæ. The results of these investigations will be published in detail elsewhere when it has been ascertained whether any breakdown of glucose occurred during the course of the experiments.

Permeability to Fluorides

The demonstration of penetration of enamel by fluorides is exceptionally difficult and, unless much more sensitive methods of micro-analysis for fluorine are developed, can only be done by the use of the radioisotope ^{18}F. Brudevold *et al.* (1957) immersed teeth in labelled fluoride solutions, washed them and then counted the surface which was subsequently polished with pumice; subsequently radioactivity suggestive of some minor penetration of the enamel by the labelled fluoride was still found on the polished surfaces. We have performed similar experiments and have obtained results identical to theirs. This experiment needs further support because during the polishing there must be a time when only a part of the surface layers has been removed. In addition, it is possible that some ^{18}F taken up in the polishing slurry may readsorb on to the freshly polished surfaces.

If we produce autoradiographs of serial sections of a tooth which has been immersed in a labelled fluoride solution and from which the surface layers have been ground away and polished in certain areas, we find an increased uptake of ^{18}F on the ground and polished enamel (Fig. 1). This experiment was performed because we wished to show that the deeper layers of the enamel, which have a low fluoride content, would take up fluoride more readily than the surface layers which have a high fluoride content. It was subsequently shown (Fremlin *et al.*, 1959) that in fact radioactive fluoride exchanges more rapidly at first with the non-radioactive fluorine of the enamel than with other groupings which will exchange with fluorides (such as hydroxyl): iso-ionic exchange occurs more readily than hetero-ionic exchange. Therefore the observed increased uptake of ^{18}F on the ground enamel could not be accounted for by the lower fluoride content of the deeper layers of the enamel. Neither could the greater uptake on the ground and polished areas of the enamel be entirely due to the production of an increased surface area because the polishing would remove surface scratches. Another possible explanation is that ^{18}F and the fluoride ions are able to penetrate the deeper layers of the enamel more quickly than the surface layers.

FIG. 1.—Autoradiograph (×1½) of two serial sections of an undecalcified premolar from which part of the surface enamel had been previously removed buccally and lingually and which had been immersed for three hours in a 2·2 p.p.m. sodium fluoride solution labelled with ^{18}F.

Note the increased uptake of ^{18}F on the ground surfaces, on the cementum and in the fissure. In the upper autoradiograph the slight activity within the root dentine suggests that the ^{18}F has penetrated through the underlying cementum towards the pulp. The lower autoradiograph of a more central section does not show a similar activity in the root dentine.

The radioactive isotope ^{18}F has great disadvantages for demonstrating penetration of the enamel by autoradiography. The short half-life of ^{18}F (110 minutes) precludes its use in experiments of long duration; indeed the dental literature contains few references to experiments carried out with ^{18}F for periods of longer than one hour. In producing autoradiographs of a tooth following its immersion in a solution labelled with ^{18}F, the ^{18}F will continue to decay while the sections of the tooth are being prepared and while the tooth is exposed to the photographic film in making the autoradiograph. Therefore if adequately exposed autoradiographs are to be prepared, the immersion in the labelled fluoride solution can only be continued for a period during which a high activity of ^{18}F remains in the hard dental tissues. With a very high initial activity of the labelled fluoride solution, it has been possible to immerse the teeth in the fluoride solution for up to nine hours and to prepare autoradiographs of satisfactory exposure of sections of these teeth.

The resolution obtainable under favourable conditions in an autoradiograph varies inversely with:

(1) The energy of the particles emitted. In the case of ^{18}F these are highly penetrating, thus limiting the possible degree of resolution very considerably.

(2) The thickness of the sections. In the case of enamel, it is only possible to prepare sections about 100 microns thick within a period of about thirty minutes. If thinner sections are prepared, considerably greater time is taken in their preparation, during which time the radioactivity will still be decreasing. The unavoidable

thickness of the sections reduces the resolution that can be obtained.

(3) The distance of the histological section from the photographic film. This factor produces no difficulties in autoradiographs of the hard dental tissues.

(4) The thickness of the emulsion of the photographic film. Fine grain films with an emulsion thickness of only 10 microns are now available and were frequently used in these experiments; unfortunately they are slow.

The difficulties arising from the high energy of the particles and the thickness of the prepared sections did not permit a degree of resolution in the autoradiographs sufficient to demonstrate convincingly any penetration of ^{18}F which occurs into intact mature enamel in a period of nine hours' immersion. Penetration into dentine and carious lesions (Fig. 2) could be demonstrated satisfactorily.

Two other methods were evolved to show penetration of ^{18}F. In pilot studies with the first method, a tooth was covered with wax apart from one relatively flat undamaged enamel surface, and immersed in a labelled fluoride solution for periods of up to six hours. Two parallel flat surfaces about 5 mm. apart were ground into this flat surface with a stone, isolating an area of unground enamel between them. This untouched surface was examined closely under the microscope and by focusing with an oil immersion lens on the surface and on the two parallel ground surfaces at either side it was possible to estimate its height above these two ground areas. The surface layers of the hitherto untouched surface were then removed by filing with emery paper and the microscope was again focused on this surface and on the two parallel ground areas at either side, enabling the new height of the central surface to be estimated. The approximate thickness of the layer removed by filing could thus be assessed. Autoradiographs were prepared of this filed surface which in certain experiments was found to be still radioactive due to the presence of ^{18}F. The danger of transference of ^{18}F from the surface layers of the enamel on to the filed surface was reduced by using fresh dry emery paper for each stroke of the filing. This set of experiments showed that penetration through sound enamel occurs to the depth of a few microns in a period of six hours.

In this experiment a positive result as shown by the autoradiograph becoming exposed over the filed surface demonstrates that ^{18}F has penetrated the enamel: a negative result shows that no penetration has occurred down to the level of the filed surface but does not necessarily show that smaller depths of penetration have not occurred.

The second method for demonstrating penetration is more complicated and needs some explanation. As ^{18}F decays, it emits positrons which, although of high energy and penetrating power compared with most beta particles, do not penetrate as far as most gamma radiations. Each positron will be stopped and be attracted to a near-by electron with which it will be annihilated, emitting highly penetrating secondary gamma rays. If ^{18}F is deposited as a surface film on a tooth, the number of beta particles and gamma rays emitted from the surface will bear a constant relation to each other (Fig. 3). If the ^{18}F penetrates appreciably below the surface of the tooth, some of the beta particles will fail to reach the enamel surface; the secondary gamma radiations

FIG. 2.—Autoradiograph (×2) of serial transverse sections of a premolar with a carious interstitial lesion which had been immersed for three hours in a 2·2 p.p.m. sodium fluoride solution labelled with ^{18}F.

Note the penetration of ^{18}F into the carious area.

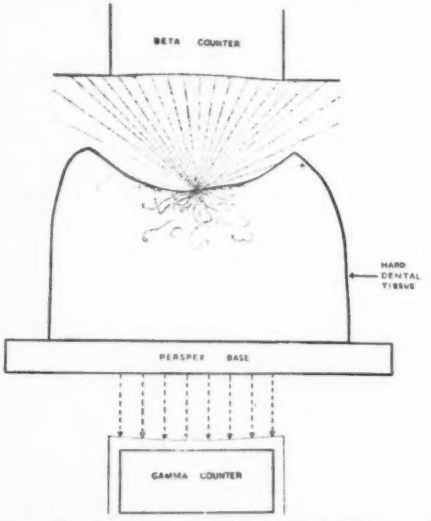

FIG. 3.—Diagram to illustrate the positrons and secondary gamma radiations emitted from a point source of ^{18}F on a tooth surface. For clarity, the positron tracks within the hard dental tissues have been magnified. Note that many positrons enter the window of the beta counter.

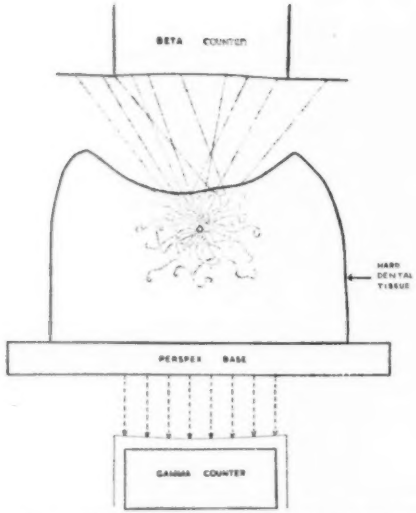

FIG. 4.—Diagram, similar to Fig. 3, in which a point source of ^{18}F lies below the tooth surface. Note that fewer positrons enter the beta counter window but that the secondary gamma radiations are not reduced.

emitted through the tooth surface will, however, not be affected significantly by the thickness of enamel in any tooth (Fig. 4). As a result, if penetration occurs, the $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ ratio will decrease and from the change in this ratio the mean depths of ^{18}F in the enamel after varying periods of immersion in the labelled fluoride solution can be compared.

The combined beta and gamma radiations can be counted satisfactorily in an end-window Geiger-Müller counter. The gamma rays can also be counted in this type of counter if the specimen is covered with a thickness of wax accurately adapted to the tooth surface and of sufficient thickness to stop all the beta particles. It should be emphasized, however, that random errors, associated with all counting procedures, will occur but the random errors arising in the counting and from the background radiations can be estimated mathematically if the number of counts on the Geiger-Müller counter is known: the greater the number of counts, the smaller are the random errors.

In this second method the crown of a recently extracted tooth was waxed on to a polythene template and covered with wax except for the enamel surface to be examined which had previously been lightly buffed to remove surface films and debris. Neither polythene nor wax takes up ^{18}F. The polythene template could be accurately positioned within a polythene retainer fixed to a tray of the Geiger counter. At the beginning of the experiment the tooth and template were placed in a fluoride solution labelled with ^{18}F. After two minutes' agitation the tooth and template were removed from the labelled solution, washed for 30 seconds under a stream of running water, dried with absorbent paper and then positioned on the counting tray. The gamma radiations and the combined beta and gamma radiations were counted separately over periods of time adequate to obtain satisfactory statistics. The tooth and template were then removed from the counting tray and re-immersed in the labelled fluoride solution for long periods which in various experiments ranged from 200–1,200 minutes during which the solutions were kept agitated. Subsequently the tooth and template were removed from the labelled solution, washed, dried and counted for gamma, and combined beta and gamma radiations as before. $\frac{\text{Beta} + \text{gamma}}{\text{gamma}}$ ratios were worked out for 78 such experiments, the necessary allowances being made for background count, the dead time of the counter, &c. The random errors were also estimated.

Attention should be drawn to the fact that this method of demonstrating penetration of enamel provides an index of the mean depth of penetration of the isotope into the whole area examined. A small defect in the enamel might allow considerable penetration in that one area which would alter the $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ ratio for the whole surface appreciably. In the enamel surfaces examined there was no clinical evidence of defects. In a few cases the specimens were sectioned and autoradiographed but no penetration of ^{18}F was shown in localized areas of these specimens. The $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ ratio does not indicate the mean depth of penetration of the isotope in microns or millimetres because it would be necessary to know also the distribution of ^{18}F at varying depths of enamel for this to be estimated. It does, however, enable the mean depth of penetration within the same enamel specimen after varying times of immersion in this solution, or the ratio of penetration in different enamel specimens, to be compared. It also enables the rates of penetration of different isotopes to be compared approximately, after calculating the effects produced by the different energies of their alpha and beta particles.

No significant differences were found between the $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ ratios at the end of two minutes' immersion and after periods of

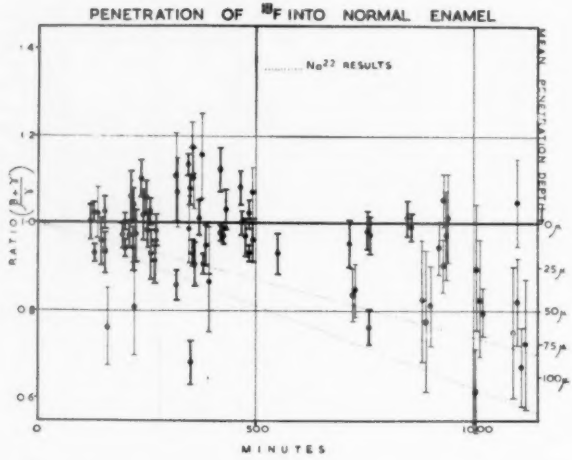

FIG. 5.—Penetration of intact enamel by ^{18}F, demonstrated by the changes in the ratios of $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ radiations emitted from a tooth surface after two minutes and after long periods of immersion in ^{18}F. *Note:* (1) The length of the vertical lines on either side of the points represents the standard deviation. (2) The scale on the right of the chart represents the increase in the mean depth of penetration of the ^{18}F, assuming a similar distribution of the ^{18}F during the second set of readings of $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ counts as in the first set at two minutes' immersion. (3) The dotted lines indicate the range of penetration of ^{22}Na which occurred when some of the enamel specimens were subsequently immersed in solutions labelled with ^{22}Na.

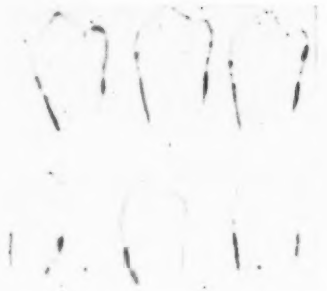

FIG. 6.—Autoradiograph (×1·5) of serial sections of a sound premolar tooth after immersion in a sodium chloride solution, labelled with ^{22}Na, for twenty-four hours. Note that the ^{22}Na has not penetrated the enamel appreciably.

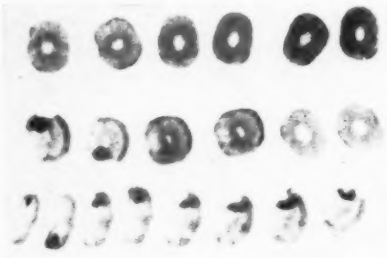

FIG. 7.—Autoradiographs (×1·5) of serial transverse sections of an incisor tooth with an interstitial carious lesion after immersion in sodium chloride solution, labelled with ^{22}Na, for fifteen days. Note that the ^{22}Na has penetrated the full thickness of the enamel, cementum and dentine and that increased uptake has occurred in the carious lesion.

FIG. 8.—Autoradiograph (×1·5) of serial transverse sections of an incisor tooth, with early carious lesions mesially and distally and with the surface layers removed from part of the buccal surface, which had been immersed for four days in a sodium chloride solution labelled with ^{22}Na. Note the increased uptake of ^{22}Na in the carious lesions and under the area from which the surface layers had been ground away (B).

immersion up to eight hours. For longer periods the ratios began to drop significantly (Fig. 5). It was therefore apparent that fluoride ions are able to penetrate enamel.

Penetration of ^{18}F into teeth of patients aged over 20 and aged under 20 was compared but no significant differences were apparent between the indices of penetration in the two groups.

Most experiments were performed on teeth extracted within four hours of the start of the experiment but some teeth were lightly polished and maintained in a humid atmosphere at 3° C. until required. The rates of penetration of ^{18}F in freshly extracted teeth and in the latter group of teeth were compared but no significant differences were found.

Permeability to Sodium Ions

Penetration of sodium in the form of sodium chloride labelled with ^{22}Na can easily be demonstrated by autoradiographic techniques. The half-life of ^{22}Na is two and a half years. It emits

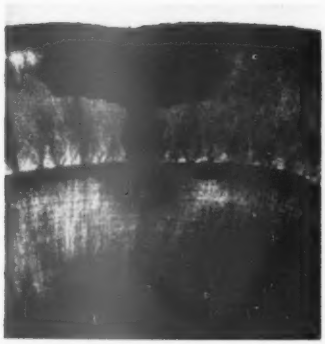

FIG. 9.

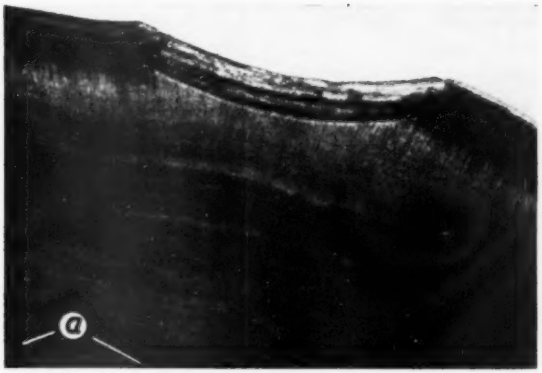

FIG. 10.

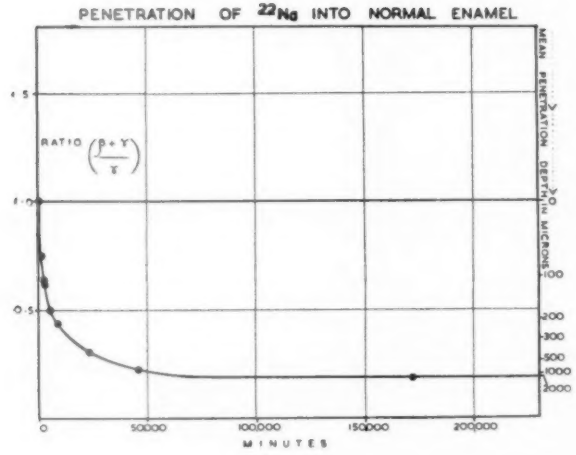

FIG. 11.

FIG. 9.—Photomicrograph (×11) of the early carious lesion, indicated by "A" in Fig. 8.

FIG. 10.—Photomicrograph (×11) showing the field indicated by "B" in Fig. 8 of the part of the buccal surface from which the surface layers have been ground away. "a" indicates the amelo-dentinal junction.

FIG. 11.—A chart indicating the depths of penetration of ^{22}Na through intact mature enamel in a tooth section about 2 mm. thick, after varying periods of immersion in a labelled sodium chloride solution.

primary gamma rays and beta particles in the form of positrons of very similar energy to the positrons of ^{18}F. In one day (Fig. 6) little penetration occurs but after fourteen days' immersion often the full thickness of the enamel and dentine is penetrated (Fig. 7). As with fluorine, an increased uptake of sodium occurs in areas of enamel from which the surface layers have previously been ground away and in carious lesions (Figs. 8, 9, and 10). As ^{22}Na emits both positrons and primary gamma radiations, penetration can be demonstrated by calculating the $\frac{\text{beta} + \text{gamma}}{\text{gamma}}$ ratios after varying periods of immersion in the labelled sodium solutions as was done with ^{18}F (Fig. 11). Comparison with the results obtained with ^{18}F suggests that the penetration of fluoride ions is slower than that of the sodium ions into intact enamel (Fig. 5). Alternatively, and more probably, the same effect would be produced by a marked adsorption of the labelled fluoride within the outermost layers of the enamel, combined with penetration of ^{18}F at the same rate as the ^{22}Na into the deeper enamel.

Conclusions.—(1) Fluoride and sodium ions and glucose molecules or products of the breakdown of glucose penetrate rapidly through carious enamel.

(2) Fluoride and sodium ions penetrate intact mature enamel; the mean depth of penetration of sodium ions increases more rapidly than that of fluoride ions.

(3) The outermost layers of the enamel are more slowly permeable to sodium ions than the

deeper layers. There is evidence suggesting a slower rate of penetration of the outer enamel by fluoride ions.

Acknowledgments.—We wish to thank Mrs. J. Mathieson and Mr. J. N. Kudahl for their help in these investigations; Dr. E. A. Marsland for the photomicrographs; and the Faculty of Medicine of the University of Birmingham and the Medical Research Council for financial assistance during the earlier and later stages of this research respectively.

REFERENCES

AITKEN, H. N. (1949) *N.Z. dent. J.*, **45**, 75.
ATKINSON, H. F. (1947) *Brit. dent. J.*, **83**, 205.
BRUDEVOLD, F., HEIN, J. W., BONNER, J. F., NEVIN, R. B., BIBBY, B. G., and HODGE, H. C. (1957) *J. dent. Res.*, **36**, 771.
FREMLIN, J. H., HARDWICK, J. L., and MATHIESON, J. (1959) *J. dent. belge*, **50**, 51.
ISAAC, S., BRUDEVOLD, F., SMITH, F. A., and GARDNER, D. E. (1958) *J. dent. Res.*, **37**, 318.
JENKINS, G. N., and SPEIRS, R. L. (1954) *J. dent. Res.*, **33**, 734.

BOOKS RECENTLY PRESENTED AND PLACED IN THE SOCIETY'S LIBRARY

Burlingame (C. C.). A psychiatrist speaks: the writings and lectures of Doctor C. C. Burlingame, 1885–1950. pp. 189. Privately printed for Mrs. C. C. Burlingame. 1959.

Dowling (H. F.). Tetracycline. pp. 64. New York: Medical Encyclopedia. 22s. 1955.

Kjer (P. V.). Infantile optic atrophy with dominant mode of inheritance. pp. 147. Copenhagen. 1959.

Lepper (M. H.). Aureomycin (Chlortetracycline). pp. 156. New York: Medical Encyclopedia. 30s. 1956.

Maia (J. de A.). Bibliografia Brasileira de Medicina. Vol. 4, 1941–1952. pp. 894. Rio de Janeiro: Instituto Brasileiro de Bibliografia e Documentação. 1958.

Pisa. Istituto di Richerche V. Baldacci. 2me Symposium de la Fondation Valentino B. Baldacci (Seteais-Lisbonne 7–11 juin 1957). Hémostase spontannée, thrombose et coagulation sanguine. pp. 214. Pisa: Omnia Medica. 1959.

Shanks (S. C.), Kerley (P. J.), and **Twining (E. W.),** eds. A textbook of X-ray diagnosis by British authors. Vol. 4, Bones, joints and soft tissues. 3rd ed. pp. 714. London: H. K. Lewis. £5 10s. 1959.

Whitby (M.). Investigations of disease. pp. 304. Chicago: Cutler Publications. 1951.

Williams (J. H. H.). Men of stress. pp. 374. London: Jonathan Cape. 15s. 1948.

Williams (J. H. H.). Between life and death. pp. 288. London: Jonathan Cape. 15s. 1951.

Williams (J. H. H.). The conquest of fear. pp. 240. London: Jonathan Cape. 16s. 1952.

Williams (J. H. H.). Don Quixote of the microscope: an interpretation of the Spanish savant Santiago Ramon y Cajal, 1852–1934. pp. 255. London: Jonathan Cape. 15s. 1954.

Williams (J. H. H.). A doctor looks at miracles. pp. 232. London: Blond. 16s. 1959.

BOOKS RECEIVED FOR REVIEW

British National Formulary 1957. Second amendment 1959. pp. 4. London: Pharmaceutical Press. 3d. 1959.

Landells (J. W.). Essential principles of pathology. pp. x+278. London: Pitmans. 25s. 1959.

Goldberger (E.). A primer of water, electrolyte and acid-base syndromes. pp. 322. London: Kimpton. 45s. 1959.

Hall (I. Simson). Diseases of the nose, throat and ear. 7th ed. pp. vi+467. Edinburgh and London: Livingstone. 21s. 1959.

Jamieson (R. A.), and **Kay (A. W.).** A textbook of surgical physiology. pp. vii+623. Edinburgh and London: Livingstone. 55s. 1959.

Medical Annual 1959. 77th issue. Edited by R. Bodley Scott and R. Milnes Walker. pp. xl+620+20. Bristol: John Wright. 42s. 1959.

Stadtsbaeder (S.). L'Auto-immunisation en pathologie. pp. 185. Brussels: Editions Arscia. 1959.

Weber (F. Parkes). Miscellaneous notes. (Third series). pp. 8. London: H. K. Lewis. 2s. 6d. 1959.

Williams (K. G.). The new frontier: Man's survival in the sky. pp. vii+161. London: Heinemann. 21s. 1959.

Winsor (T.). Peripheral vascular diseases. pp. xviii + 845. Springfield, Ill.: Thomas; Oxford: Blackwell. £6 5s. 1959.

Zimmerman (H. A.), ed. Intra vascular catheterization. pp. xviii+782. Springfield, Ill.: Thomas; Oxford: Blackwell. £6 6s. 1959.

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MEETING AT ST. ANN'S HOSPITAL, LONDON

THE programme was as follows:

Demonstration and Discussion of Manipulative Methods in Physical Medicine.—Dr. W. HARGRAVE-WILSON (*see E. Afr. med. J.*, 1950, **27,** 396; 1953, **30,** 377).

Respiratory Poliomyelitis: Introduction by Dr. G. DONALD W. MCKENDRICK. Cases were shown.

Other cases were shown by Dr. J. R. ELLIS, Dr. I. C. GILLILAND, Dr. P. B. CROFT, Dr. J. H. GLYN and Mr. E. H. HAMBLY, as follows:

(1) **Rheumatic Fever with *Salmonella typhimurium* Urinary Infection.**

(2) **Scleroderma with Family History.**

(3) **Gout and Addison's Disease.**

(4) **Spinal Cord Cyst** (histology not available).

(5) **Ankylosing Spondylitis and Psoriasis.**

(6) **Primary Tumour of Patella** (*see* HAMBLY, E., 1959, *Proc. R. Soc. Med.*, **52,** 576).

(7) **"Yaws" of Fibula** (*see* HAMBLY, E., 1959, *Proc. R. Soc. Med.*, **52,** 575).

Meeting
March 11, 1959

DISCUSSION ON THE PAINFUL SHOULDER

Dr. Geoffrey O. Storey (London):

The purpose of this communication is mainly to discuss the differential diagnosis of the painful shoulder, but first it is necessary to make some definitions.

Most of the conditions which cause pain in the shoulder may be associated with a stiff shoulder and therefore in discussing the causes of pain I am discussing the ætiology of the stiff shoulder. By stiff shoulder I mean that condition which has had various names, i.e. capsulitis, periarthritis, frozen shoulder. I believe that supraspinatus tendinitis and rotator cuff lesions are forms of the same syndrome and I do not intend to subdivide them. The causes of pain in the shoulder are: (1) Those which lie distant from the shoulder and cause pain of a referred type. (2) Those which lie in and around the shoulder-joint.

Lesions Distant from the Shoulder

In the first group of conditions are those which affect the diaphragm. This structure develops embryologically in the neck and migrates distally taking with it the phrenic nerve C3, 4, 5. Therefore conditions which irritate the central portion of the diaphragm may cause referred pain in the shoulder. Below the diaphragm subphrenic abscess, liver abscess and cholecystitis are some of many conditions which might be mentioned but these are not often met in a Department of Physical Medicine. The upper surface of the diaphragm may be irritated by diaphragmatic pleurisy and those conditions which cause this.

A stiff shoulder is not often associated with these diaphragmatic conditions but this association is sometimes noticed with the intrathoracic causes of pain in the shoulder. The findings in a series of 67 patients with stiff shoulders, selected by having pain and limitation of movement of the shoulder and therefore excluding less severe variations of the syndrome, are shown in Table I (age and sex distribution) and Table II (associated clinical conditions); it will be noted that there is only one patient under 40. The classification of the associated conditions was compiled on a clinical basis; further analysis would probably result in a further breakdown of cases with no obvious associated condition (fractures of the humerus and dislocation of the shoulder are excluded).

TABLE I.—AGE INCIDENCE—67 CASES

30–40	40–50	50–60	60–70	Over 70
1	17	15	19	15

Male 32; Female 35.

TABLE II.—ASSOCIATED CONDITIONS—67 CASES

Nil	17
Trauma	23
Colles' fracture	3
Cardiovascular disease	10
Chest disease	3
Osteoarthritis of shoulder	2
Cervical spondylosis	3
Hemiplegia	3
Herpes zoster	1
Psychological factors	6

Cervical spondylosis refers to clinical as well as radiological cervical spondylosis. Osteoarthritis of the shoulder refers to gross radiological osteoarthritis of the gleno-humeral joint. Psychological factors appeared to be important in 6 patients; the abnormal findings ranged from one patient with a florid hysteria to others with anxiety states and compensation neurosis. It should be noted that some writers believe that the stiff shoulder is a psychosomatic disorder. A "periarthritic personality" has been described which is said to be particularly prone to this disorder (Coventry, 1953). The patients are described as passive and apathetic; they expect someone else to get them well and would rather be massaged than do active exercises. "Poised indecisiveness" has been used to describe their attitude. While I have not been impressed by any particular types being affected with this condition, nevertheless it is clear that in some cases psychological factors are very important.

The patients with intrathoracic lesions are further analysed in Table III. Two of those with

TABLE III.—INTRATHORACIC LESIONS

Cardiac disease		10 cases
Cardiac pain	6	
Auricular fibrillation	6	
Mitral stenosis	2	
Aortic stenosis	1	
Chest disease		3 cases
Apical tuberculosis—lobectomy	1	
Carcinoma of lung—thoracotomy	1	
Spontaneous pneumothorax	1	

lesions other than cardiac had surgical procedures; the patient with spontaneous pneumothorax was important since there was a close time relationship between the onset of pneumothorax and the development of the stiff shoulder. Although these and other intrathoracic lesions may sometimes be associated with a stiff shoulder, this association has been most frequently observed with cardiac lesions, particularly with cardiac pain, and this merits further consideration.

Pain impulses arising in the heart must pass from the heart via the sympathetic system and enter the central nervous system in the first four thoracic segments. Cardiac pain, therefore, is felt mainly in the area supplied by these sensory nerves but the pain is often referred over a much wider area. Froment and Gonin (1956) have examined some of the more atypical radiations of cardiac pain and they believe that the pain is felt in a previously painful area. Thus a patient with cervical spondylosis feels the pain in the neck and arms, and a patient with a gastric ulcer feels cardiac pain in the epigastrium and, in the present context, patients with degenerative changes in the shoulder will feel their pain in the shoulder. These theories are in line with some of those of the mechanism of referred pain.

In addition to the sympathetic nervous system acting as afferent fibres there is also evidence of a disturbance of the sympathetic system in the area where the pain is felt. Doret and Ferrero (1951) have shown that there is a fall in temperature in this area after a cardiac infarct. Patients also describe sensations in this area which they distinguish from the cardiac pain and describe a feeling of coldness and a sensation of draught on the left side. Sensations of this sort sometimes precede typical cardiac pain and a stiff shoulder may also do this. The relief obtained in some cases of both stiff shoulder and severe cardiac ischæmia by stellate ganglion block is further evidence incriminating the sympathetic system in the causation of the stiff shoulder and in the path of referred cardiac pain.

The changes in the hand which occur in some cases of stiff shoulder seem to be due to dysfunction of the sympathetic system—the shoulder-hand syndrome (Steinbrocker, 1947). This syndrome has been particularly related to cardiac pain (Kehl, 1943; Johnson, 1943) but is seen in other conditions; in this series the most typical example was seen in the man with carcinoma of the lung. The changes in the hand consist of swelling, redness and pain leading to atrophy, scleroderma-tous changes, fibrosis in the palm and Sudeck's atrophy. These changes seem to be vascular in origin and initiated by the autonomic system and it is argued that the changes in the shoulder are also the result of sympathetic dysfunction (reflex sympathetic dystrophy) (Steinbrocker *et al.*, 1948). The alternative view that the changes in the hand are due to immobility related to the stiff shoulder is, I think, untenable. The findings in the hand in the present series are illustrated in Table IV.

TABLE IV

Hand symptoms		31 cases
Brachial neuralgia	21	
Swelling, stiffness, etc.	10	
Hand signs		12 cases
Swelling	4	
Colour change	3	
Fibrosis	7	
Contracture	1	

Brachial neuralgia is seen to be a common symptom in patients with a stiff shoulder. This may either be regarded as referred pain from the shoulder or as arising from the cervical spondylosis which may also be responsible for the stiff shoulder.

Pain in the shoulder and down the arm may, of course, also be caused by other lesions in the cervical region. These include lesions within the spinal canal, other lesions of the cervical vertebræ themselves and lesions in the root of the neck including neoplasms of the apex of lung. Cervical spondylosis has been regarded not only as a cause of pain in the shoulder but as a cause of stiff shoulder. Nevertheless there were only three cases which had clinical cervical spondylosis (i.e. stiff neck) in this series although a large number would have radiological cervical spondylosis in a series of this age distribution. It is also true that pain may be referred up the arm from lesions in the hand and forearm.

Lesions Round About the Shoulder

Any form of arthritis may affect the shoulder-joint but not many are common. Rheumatoid arthritis is the commonest and the shoulder may be the first joint affected. Ankylosing spondylitis may affect the shoulder following involvement of the spine. A Charcot's joint may be seen in the shoulder, particularly in syringomyelia. Tuberculosis involving the joint or upper end of the humerus often presents difficulty in diagnosis. This diagnosis should always be remembered, particularly in young people with a stiff shoulder, as radiological changes may not appear for some time. Tuberculosis may also occur in a joint already affected by rheumatoid arthritis. Osteomyelitis of the first rib may result in a pain in the shoulder; tumours may occur around the shoulder-joint, sarcoma of the upper end of the humerus is sometimes seen, secondary carcinoma of the lung or breast may be seen in this region.

The importance of degenerative changes in and around the shoulder-joint in the causation of the stiff shoulder has been debated. Di Palmas *et al.* (1950) have shown that degenerative changes occur as early as the third decade. In the glenohumeral joint these changes mainly involve the humeral side of the joint. Synovial changes follow with tears in the capsule and calcification in the tendons around the joint. Degenerative changes also occur early in the acromio-clavicular joint and the relationship of the joint to the tendon of the supraspinatus may be one factor in causing injury to this tendon.

These facts associated with the age incidence of the stiff shoulder and the importance of trauma suggest that the stiff shoulder is mainly a degenerative condition aggravated by trauma. The fact that radiological changes are often slight has contributed to a reluctance to make the diagnosis.

Finally one condition, acute subdeltoid bursitis, must be mentioned which, although it may be traumatic in origin, is often obscure in its cause. This condition with its sudden onset and deposition of calcium often responds well to hydrocortisone.

REFERENCES

COVENTRY, M. B. (1953) *J. Amer. med. Ass.*, **151**, 177.
DI PALMAS, A. F., WHITE, J. B., and CALLERY, G. (1950) *Amer. Acad. orthop. Surg., instr. Course Lect.*, **7**, 168.
DORET, J. P., and FERRERO, C. (1951) *Cardiologia*, **9**, 80.
FROMENT, R., and GONIN, A. (1956) Les Angors Coronariens Intriqués. Paris.
JOHNSON, A. C. (1943) *Ann. intern. Med.*, **19**, 433.
KEHL, K. C. (1943) *Ann. intern. Med.*, **19**, 213.
STEINBROCKER, O. (1947) *Amer. J. Med.*, **3**, 402.
——, SPITZERN, N., and FRIEDMAN, H. H. (1948) *Ann. intern. Med.*, **29**, 22.

Dr. P. Hume Kendall (London):

The Management of the Deranged Sub-acromial Joint

The conservative treatment of the soft tissue lesions that occur around the shoulder-joint has been a controversial subject for a very long time. Treatment has not, in the past, been directed at a specific lesion: measures are largely carried out for signs and symptoms, particularly those of pain, limitation of function and muscle spasm. Rest, physical therapy in its various forms, manipulation and the local injection of a wide variety of drugs have been recommended by various authors, the majority of whom have advocated one to the exclusion of the others and certainly without due consideration of the pathological changes involved.

At the present time, the surgical treatment of the more gross lesions of the shoulder-joint is now on a sound basis, largely as a result of the outstanding work of Duplay (1872), Codman (1909) and McLaughlin (1945) in describing the anatomical and gross pathological changes that occur in the periarticular structures and the repair thereof.

The medical treatment of these musculotendinous lesions has not been so well defined and it is only in recent times that the implications of the twin pathological entities—inflammation and degeneration—have been fully accepted. Available for use in the treatment of this group of conditions are the steroids, both systemic and by local injection, physiotherapy in its various forms, manipulation and a wide range of local analgesics. Which, if any, are justifiable procedures and how do they fit into the pathological picture?

Steroid therapy.—The most important of all these measures and by far the greatest single advance made in the treatment of shoulder lesions within the past fifty years is the introduction of local steroid injection therapy. Even this statement, although it will be accepted by the majority, will be contradicted by some.

Indeed, we find that Murnaghan and McIntosh (1955), in a controlled trial on 51 patients, did not find any significant difference between the local injection of hydrocortisone and procaine. Their technique, however, was open to question in several directions. Kuipers (1954) considered that the results of local steroid injection were inferior to systemic administration. Glyn and Newton (1958) also disclaimed the value of local hydrocortisone injections in cases of established periarthritis of the shoulder.

When the place of systemic steroids is examined, there is again controversy; Coventry (1954) did not agree that there was any advantage to be gained by their use. Thus, there still appears to be a need to justify the use of steroids in the treatment of shoulder lesions. This may be attempted in two main ways: (1) By examination of the pathological changes, (2) By recognition of the changing character of the incidence of shoulder lesions.

The first of these is the pathological approach. Glyn and Newton (1958), in their excellent review of steroid therapy, stated "from a therapeutic point of view, local steroid injection therapy is a field in which empiricism has triumphed over science". This is open to question.

Bosworth (1941) and McLaughlin (1951) have pointed out that a large number of individual tendinous and ligamentous lesions may occur within the musculo-tendinous structure and give rise to a typical supraspinatus picture. These tendinous tears usually occur in the supraspinatus, infraspinatus and subscapularis tendons close to their insertion. These defects are forced beneath the acromion or coraco-acromial ligament thus resulting in friction. Sooner or later, the bursa becomes the seat of an inflammatory process with the development of proliferative folds which pass only with difficulty below the acromion and falciform edge of the acromial ligament. The next stage is the development of an acute generalized non-specific inflammation of the whole subacromial bursa and joint and the stage is set for a "frozen shoulder".

The work of Neviaser (1945) amplifies the importance of these changes still further. In a series of studies undertaken to clarify the pathology without reference to treatment, he showed that the essential microscopical picture was that of inflammatory changes varying from acute polymorphonuclear infiltration of the capsule to a more diffuse chronic process. There was definite evidence in some of reparative inflammatory changes. These lesions are typical examples of non-specific inflammatory disease that is so eminently suited to treatment with steroids, either systemic or local. The mere fact that the more acute forms respond dramatically, and the more chronic forms less satisfactorily, is a further indication of the degree of inflammatory change that is occurring within the joint and demonstrates quite clearly that there is a series ranging from acute inflammation to chronic fibrosis with varying degrees of both occurring at every stage.

My only original contribution here is to suggest that there is a very definite change in the way in which shoulder lesions are presenting. Each year at Guy's Hospital, about 140 patients with shoulder lesions attend the Physical Medicine Department. In 1953, the year before the wide-scale introduction of local steroid injection therapy, 23% of the patients presented with "frozen shoulders". In 1956, this figure had dropped to 15%. In the past two years this group of lesions only accounted for 4–5% of the total number. This change in character of the form of presentation is, in my opinion, directly the result of the widespread use of steroids at the appropriate time.

Physiotherapy.—The value of the various forms of physiotherapy commonly prescribed is an extremely topical subject at the present time. Few will deny the accuracy of the conclusions drawn by Hamilton *et al.* (1959), that there is little place for any form of heat in the treatment of arthritis. Furthermore, in the light of the foregoing description of the pathological changes that occur around the shoulder joint, it is difficult to see just what effect such treatments can exert. On the other hand, rest and exercise therapy in its various forms has an extremely valuable part to play. In the acute forms of capsulitis or in a very acute supraspinatus tendonitis. where the inflammatory reaction is at its peak and pain and œdema at their maximum, absolute rest of the joint is essential. No matter what the age of the patient, I feel that the shoulder can be immobilized safely for periods of up to four weeks, provided that full use is made of steroids and active exercises subsequently. As the inflammatory changes subside a progressive range of movement should be encouraged, the great skill of the experienced physiotherapist being utilized to graduate movements carefully from gentle passive

movements, then active assisted, and finally fully active mobilizing exercises. Careful observation is, however, necessary to ensure that an acute inflammatory reaction is not precipitated. It is at this stage that repeated local steroid injections or even systemic steroids as a short-term measure can be exhibited with advantage.

Whilst discussing the part to be played by exercises in this group of conditions, it is worth while pointing out that with careful instruction in the exercises and occasional supervision by a competent physiotherapist, together with suitable illustrated charts, the patient of average intelligence can be encouraged to perform such exercises at home.

Manipulation.—For many centuries, manipulation has played a prominent part in the treatment of the so-called "frozen shoulder". Periarticular adhesions and capsular thickening and contractions frequently cause a limitation of movement, particularly in external rotation and abduction. It is for this reason that those who advocate manipulation, which after all is no more than a forceful passive movement of the joint, suggest that the shoulder should be moved through as full a range of movement as possible, including those in which there is a complete restriction. McLaughlin (1951) has shown, however, by manipulating a series of shoulders under direct vision through an anterior incision, that these "cracks" of adhesions breaking are no more than the following disruptions occurring within the musculo-tendinous cuff: (1) The rupture of the long head of the biceps just proximal to the bicipital groove, (2) The transverse rupture of the subscapularis tendon, (3) The tearing of the gleno-humeral ligaments from the scapula to permit external rotation, (4) Diffuse tearing and hæmorrhage occurring into the stretched tissues.

At no time did he observe any rupture of intra-articular adhesions, and in fact no one who has approached the joint surgically has ever demonstrated such adhesions. McLaughlin therefore quite rightly questions the advisability of manipulation—a sentiment reflected by Watson-Jones (1957) who stated that many shoulders become frozen as a result of unduly forceful manipulative procedures, and that the sero-fibrinous exudate of the manipulation gave rise to still more stiffness . . . from periarthritis and periarticular adhesions. It would appear therefore that this procedure, which results in such a mutilation of the structures supporting the joint, can scarcely be countenanced.

Occasionally, however, it may be justifiable, where it is essential to mobilize the shoulder of an otherwise disabled wage earner, who for economic reasons is prepared to accept the concomitant risks. On these occasions, the procedure should be carried out under full steroid cover. Steinbrocker *et al.* (1953) were among the first to point out the value of intravenous ACTH before and after manipulation. At the West London Hospital, Fearnley (1959) had a limited amount of success by manipulating the completely fixed shoulders of patients who have been prepared by the administration of cortisone. At Orpington on the few occasions that we have used this procedure, the patients have been given prednisone 40–50 mg. daily, for forty-eight hours prior to the operation. This dose must be subsequently continued for at least fourteen days, being gradually "tailed off".

I would conclude with a plea for a more rational approach to the problem of the painful shoulder. Correct diagnosis is the basis of successful treatment, and such treatment must depend upon the consideration of the relative merits of all the factors mentioned above; the patient must not be condemned to "yet another course of heat and massage".

REFERENCES

Bosworth, D. M. (1941) *J. Amer. med. Ass.*, **117**, 422.

Codman, E. A. (1909) *Publ. Mass. gen. Hosp.*, **2**, 521.

Coventry, M. B. (1954) *Proc. Mayo Clin.*, **22**, 58.

Duplay, S. E. (1872) *Arch. gén. Méd.*, **20**, 513.

Fearnley, M. E. (1959) Personal communication.

Glyn, J. H., and Newton, D. R. L. (1958) *Ann. phys. Med.*, **4**, 176.

Hamilton, D. E., Bywaters, E. G. L., and Please, N. W. (1959) *Brit. med. J.*, i, 542.

Kuipers, R. K. W. (1954) *Ned. Tijdschr. Geneesk.*, **98**, 1558.

McLaughlin, H. L. (1945) *J. Amer. med. Ass.*, **128**, 563.

—— (1951) *Bull. Hosp. Jt. Dis.*, **12**, 383.

Murnaghan, G. F., and McIntosh, D. (1955) *Lancet*, ii, 798.

Neviaser, J. S. (1945) *J. Bone Jt. Surg.*, **27**, 211.

Steinbrocker, O., Neustadt, D., and Lapin, L. (1953) *J. Amer. med. Ass.*, **153**, 788.

Watson-Jones, R. (1957) *Proc. R. Soc. Med.*, **50**, 137.

Dr. D. R. L. Newton (London):

The painful shoulder should appear high on any list of conditions for which a great deal of valueless treatment is prescribed. The earnest desire of physicians, surgeons and physiotherapists to do something to help is unfortunately not always matched by their ability to consider what exactly they are trying to achieve. Many would-be helpers are doomed to failure before they begin because they fail to differentiate between the

various common shoulder disorders and lack the knowledge necessary for the application of effective remedies in any particular case. Others feel that they have done their duty when they have provided some sort of diagnostic tag, paying no heed to the symptoms and physical signs which may be present, and making no attempt to understand the differing natural histories of these conditions. Consequently liberal supplies of drugs, injections and physical treatments are ordered and the one which happens to be in use at the time improvement occurs is hailed as the winner. Alternatively the patient may be assured, without further explanation, that there is nothing useful to be done for a painful shoulder and advised to suffer in silence.

A number of common pitfalls in the management of the painful shoulder appear to arise as the result of failure to recognize that definite clinical patterns occur, depending on varied factors such as age, sex, occupation, and duration and mode of onset of symptoms. From the history it is often possible to forecast the physical findings and the results of investigations and also to give an accurate prognosis, provided that there is an awareness of the natural histories of the various pathological conditions which occur in this region.

Typical pain of shoulder origin is never paræsthetic in quality, frequently radiates widely, certainly to the forearm and wrist and, if severe, to the fingers, but is seldom felt between the shoulder blades. It is nearly always worse at night, and is usually aggravated by lying on the affected side. A statement that pain is related to arm movement should never be accepted at face value because this may occur in a patient with a cervical spinal lesion who unequivocally has no shoulder disorder. Because the upper limb is largely suspended from the neck, the shoulder cannot perform normally unless the neck is stabilized. Stability of the neck is dependent on active mechanical processes involving the cervical articulations, and it is an abnormality there which gives rise to the pain referred to and felt in the arm. Although the relationship is less direct than he thinks the patient is correct in attributing his pain to arm movement, but the examiner should not be hoaxed too. Only firm passive support of the neck while shoulder movements are being tested will eliminate this bogus physical sign.

The curious behaviour of the disordered shoulder can only be explained by the fact that as a joint the shoulder is anatomically and functionally unique. In no other large joint is the integrity of the capsule so important, and this is dependent upon the smooth functioning of those controlling tendons which together have come to be called the musculo-tendinous, or rotator, cuff. A great deal of importance has always been attached to the function of the supraspinatus in the initiation of gleno-humeral abduction, but not enough attention is generally given to the part this and the adjacent tendons play in constantly stabilizing the humeral head. In few other joints are quite so many muscles required to act in unison in order to produce a smooth pattern of movement from any given starting point. For this reason any small defect of fixator action is apt to be reflected in an abnormal rhythm of scapulo-humeral movement, and may be felt by the patient as a varying degree of disability. In everyday life, except when complete relaxation occurs during sleep, the humeral head is almost constantly being actively stabilized so as to enable the forearm and hand to perform some function or other. However small and insignificant a movement is being carried out, its successful performance depends on perfect control of the gleno-humeral joint, a point well illustrated by the case of a man suffering from an isolated shoulder-girdle neuritis, a so-called neuralgic amyotrophy. Electromyographic examination revealed considerable denervation in the deltoid muscle. Although the supraspinatus did not appear to be wasted and no fibrillation was present on needle electrode exploration at rest no volitional motor unit activity could be found in this muscle on attempted abduction of the arm, yet when the patient was asked to flex his elbow a complete motor unit pattern appeared in the supraspinatus. It was then found that a response of considerable magnitude could also be obtained when he clenched his fist, even if his arm remained resting by his side. Voluntary attempts by the supraspinatus to participate in abduction of the arm were apparently completely inhibited by the recent memory of severe pain. It seems that we may commonly underestimate the amount of activity involved in the fixation of the humeral head, activity which appears to be a highly important factor in the pathogenesis of some common shoulder disorders.

Confusion is caused by the varied terminology applied to the painful shoulder. Until universal agreement is reached on the exact relationship between pathology on the one hand and symptoms and physical signs of disordered function on the other, we should describe what we actually observe and put the diagnostic tags into inverted commas. It is no use making a diagnosis of say painful shoulder, frozen shoulder, capsulitis, or peri-arthritis, unless we define accurately what we mean in terms of altered function, because all these names may indicate the same thing to some people while meaning quite different things to others. Only by carefully defining the terms we

use can we make true comparisons between one group of cases and another, and at least one recent effort to perform a controlled clinical trial was largely invalidated by the lack of definition of the term "painful shoulder" (Newton, 1955). It is inconceivable that anyone with experience in this field believes that all cases behave in a comparable way, irrespective of the history and physical findings.

Over the years the complex mechanism of shoulder movement has been studied by both anatomists and clinicians, but knowledge is of little use unless it is applied when things go wrong. Every component that goes to make up the complicated pattern of movement should be separately assessed wherever possible, so that the disorder of function can be correctly evaluated. First, the relationship between gleno-humeral and scapulo-thoracic movement must be determined: then a careful assessment of what is possible on active movement should be compared with what can be achieved passively: and finally, individual movements must be tested against resistance. To carry this scheme to a successful conclusion does not involve one in any discussion concerning the nature of the pathological changes present, but merely increases the chances of indicating in which structure the disorder of function lies. To this information can be applied acceptable pathological concepts which, when taken together with a knowledge of the natural history of the various patterns of disorder, will provide most of the information necessary to the solution of the individual problem.

There is little that is controversial about the pathology of the shoulder in such conditions as tuberculosis and suppurative arthritis, but the vast majority of shoulder lesions with which we have to deal appear at first sight to have no specific ætiological factor. However, the basis of nearly all these conditions is an admixture of degeneration and trauma, the proportion of each varying from case to case. The almost continual use of the musculo-tendinous cuff seems to explain why a clear-cut history of trauma is so often absent even in the older age groups. The repeated small traumata applied to the supraspinatus tendon of a housewife, who in addition to house and children also has to contend with a job, may be quite as effective a stimulus as the jerk her unmarried sister may get when she attempts to jump on to a moving bus. Grant and Smith (1948) have demonstrated that degeneration in the upper part of the capsule increases in arithmetical progression as age advances. There is also evidence that not inconsiderable degenerative changes may occur without the production of symptoms, and Harrison (1949) has shown that actual tears in the structure of the musculo-tendinous cuff have occurred in the absence of significant symptoms. In this context the term "significant symptoms" provides us with an important key to the understanding of these disorders. First there is the example of the anxious individual with a low pain threshold who may be thought to be suffering from so-called psychogenic rheumatism because cursory examination has proved entirely negative. However, careful history-taking reveals an organic glint shining through the mist of neurotic manifestations, and careful examination indicates that minor, but significant, physical signs do in fact exist. The matter can often be dealt with in a few minutes by the use of a little hydrocortisone, and the reassurance which is still required will now successfully achieve its object. The second example is provided by the stoic who will put up with a lot of discomfort without complaining about what he regards as the ordinary aches and pains of life. In the first case the doctor is too blind to see, and in the second case is not given the opportunity to discover, the manifestations of degenerative/traumatic lesions in the early stages of their development: but this is not to say that they do not occur, and the more diligently we inspect the patient and his history the more frequently do we find them. The pure neurotic or the malingerer who presents with shoulder pain invariably overplays his hand, and both the history and the pattern of abnormalities which become manifest from careful examination are quite unlike those which commonly occur in association with even a mild organic lesion. Conversely, nobody with a normal shoulder can successfully mimic an abnormal scapulo-humeral rhythm, so that this finding invariably means that there is at least some organic basis for the patient's symptoms.

One sees every stage of development of these traumatic/degenerative lesions ranging from the minimal supraspinatus or infraspinatus tendinitis right through to the ultimate lesion, the classical, fully developed capsulitis, periarthritis or frozen shoulder, call it what one will, with its attendant severe pain and limitation of all movements. We have all occasionally converted a localized lesion of the musculo-tendinous cuff into a frozen shoulder by means of enthusiastic but misguided over-treatment. Although most people now seem agreed on the importance of rest in the early stages we sorely need to know what are the precise factors which bring about this change. A chronic or recurrent case of "supraspinatus syndrome", which even after some years has remained strictly localized, may, for no apparent reason, suddenly develop into a frozen shoulder, while other, apparently similar, cases settle down spontan-

eously. In other instances minor symptoms occur with minimal physical signs, and yet however much or however little we do a frozen shoulder steadily develops.

The term "frozen shoulder" should be reserved solely for that inexorable march of events wherein there is increasing pain and decreasing movement, later to be replaced by a stiff and more or less painless gleno-humeral joint which ultimately regains a useful, if not full, range of movement. The term "capsulitis" should be used to convey the idea that an inflammatory process of some kind has spread beyond the confines of one of the tendons forming the musculo-tendinous cuff to involve the capsule and its environs producing limitation of gleno-humeral movement, but complete involvement of the capsule need not necessarily occur and by no means every case of capsulitis develops into a frozen shoulder. The limitation of movement at the gleno-humeral joint is usually of that pattern in which external rotation is reduced most and internal rotation least, with abduction somewhere between the two extremes. Simmonds (1949) points out that the pattern of limitation of movement usually depends on the position in which the shoulder is rested.

Degeneration is not the only pathological process occurring in the region that conditions the musculo-tendinous cuff to the development of a painful lesion. In the presence of rheumatoid arthritis or ankylosing spondylitis it is very easy to accept a painful shoulder at face value, that is, as being due to rheumatoid-type changes in the shoulder-joint itself. Proper assessment of shoulder function will often show that there is, in fact, little or no limitation of passive gleno-humeral movement, but that the symptoms arise partly or wholly from a musculo-tendinous cuff lesion, and this may respond dramatically to the proper treatment. The common occurrence of these tendinous and bursal lesions at the rheumatoid shoulder is analogous to those described by Brewerton (1957) as occurring in the hand and by Burt (1958) as occurring in the foot and ankle.

Sometimes an apparently innocent shoulder capsulitis may not in fact be the simple outcome of degeneration plus trauma that it at first appears. A frozen shoulder, using the term in the sense that has already been indicated, occurs not infrequently in association with a grossly raised E.S.R. but without any coincident systemic disease, and seems to have a predilection for elderly males. The condition may occur bilaterally, when one shoulder usually lags behind the other by a matter of a week or two. Most cases observed by the author have run a benign course ending after one or two years with a useful, if not full, range of gleno-humeral movement, and the differential agglutination titre has always remained negative. In this group may be placed a man of 66 with recent rheumatoid involvement of the small joints of the hands and feet, this having been preceded two years before by a frozen shoulder of the type described. Whatever the age of onset, therefore, this type of case should be kept under review for some years, and the disorder should always be regarded as the first manifestation of rheumatoid disease until proved otherwise. This curious shoulder arthropathy is, incidentally, one of the conditions which some authors grace with the somewhat misleading title of infective arthritis.

Finally, there is the fascinating problem posed by what may be termed pseudo-shoulder signs. Inman and Saunders (1944) have drawn attention to the fact that sclerotogenous pain is accompanied by local tender spots situated at the insertion of tendons into bones. Although we may differ in the significance which we attach to them, we are all aware of the referred tender spots which commonly occur around the shoulder girdle in the presence of degenerative cervical spinal disease. Many patients present with typical shoulder type pain and with the physical signs of a musculo-tendinous cuff lesion, but seem extraordinarily resistant to treatment. Apart from minor clinical signs in the neck and the well-marked radiological changes of cervical spondylosis no other lesion can be detected locally or systemically. Empirical treatment applied to the neck, such as traction or immobilization in a collar, may produce a rapid disappearance not only of symptoms referred from the neck but also of the shoulder signs. Consideration of this type of case, taken in conjunction with those of the pseudo-tennis-elbow variety, which follows a basically similar pattern, suggests that conditioning of tendons may be mediated through nervous pathways, possibly via the autonomic system. It seems as if some alteration in physical state may occur in structures within the same sclerotome as that in which the primary lesion is sited, and that this makes them unusually vulnerable to the stress and strain of everyday function.

REFERENCES

BREWERTON, D. A. (1957) *Ann. Rheum. Dis.*, **16**, 183.

BURT, H. A. (1958) *Proc. R. Soc. Med.*, **51**, 255.

GRANT, J. C. B., and SMITH, C. G. (1948) *Anat. Rec.*, **100**, 666.

HARRISON, S. H. (1949) *J. Bone Jt. Surg.*, **31** B, 418.

INMAN, V. T., and SAUNDERS, J. B. (1944) *J. nerv. ment. Dis.*, **99**, 660.

NEWTON, D. R. L. (1955) *Lancet*, ii, 875.

SIMMONDS, F. A. (1949) *J. Bone Jt. Surg.*, **31** B, 426.

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Section of Medicine

President—A. H. Douthwaite, M.D., F.R.C.P.

Meeting
April 28, 1959

DISCUSSION ON MEDICAL DISEASES IN PREGNANCY

Dr. Morgan Jones (Manchester):

Heart Disease in Pregnancy

This account cannot cover, even in outline, all the problems which arise when a pregnant woman has heart disease. The topics selected are those in which we have been particularly interested in recent years. One of the difficulties inherent in the study of this subject is that the risks of death or serious complication are comparatively slight and it is quite possible to see 100 consecutive cases without any important complications of any kind. Consequently a large series of cases is essential if a representative picture is to be drawn.

Heart failure in pregnancy.—The central problem of heart disease in pregnancy is the incidence, treatment and prognosis of heart failure. 90% of heart disease in pregnancy is rheumatic, usually with mitral stenosis. In rheumatic heart disease either right ventricular failure with dependent œdema or acute pulmonary œdema may occur, and we have been interested in the relative frequency and prognosis of these two conditions. In 1,000 consecutive pregnancies (Table I) right heart failure occurred in 68 instances, and 9 of these patients died during the pregnancy or within eight months of delivery, a failure mortality of 13%, and an overall mortality of 0·9%. In the same 1,000 pregnancies 27 instances of acute pulmonary œdema occurred, 11 of these patients died; a failure mortality of 41% and an overall mortality of 1·1%. The total mortality from heart failure in pregnancy has been 2% in these 1,000 cases. Pulmonary œdema occurs much less than half as frequently as right heart failure, but when it does occur the mortality is more than three times as great, and rather more patients die from this than from ordinary congestive heart failure. The much better prognosis of right heart failure is an indication of the greater effectiveness of treatment in this condition during pregnancy.

Table I.—Heart Failure in Pregnancy (1,000 Pregnancies)

	Congestive heart failure	Pulmonary œdema
Number	68	27
Deaths within eight months postpartum	9 (13%)	11 (41%)
Overall mortality	0·9%	1·1%

There are other significant differences between these two types of failure. First the type of heart disease is different. Patients who develop right heart failure were usually considerably incapacitated before pregnancy, often have large hearts and myocardial damage may be an important contributory factor, whereas those who develop acute pulmonary œdema may have had little in the way of symptoms before pregnancy, may have hearts which are not enlarged and often have isolated mitral stenosis without appreciable myocardial damage. In assessing fitness for pregnancy, it has been the custom to attach a great deal of weight to the patient's capacity for effort before pregnancy and this is still a good guide to the likelihood of right heart failure, but it is not a reliable means of assessing the likelihood of acute pulmonary œdema, for this may develop in patients with slight or no symptoms before pregnancy. The fact that more patients actually die from acute pulmonary œdema than from right heart failure emphasizes the need to introduce new criteria in assessing fitness for pregnancy, and the problem of patients who are well before pregnancy but run into considerable trouble during pregnancy will be referred to later.

Nor are these the only differences between right heart failure and acute pulmonary œdema in pregnancy. The work of Hamilton and Thomson (1941) showed that the onset of right heart failure occurred most often around the 28th to 36th weeks of pregnancy, when the hæmodynamic changes of pregnancy reached their peak. This remains an acceptable general guide, and it is very uncommon for right heart failure to develop for the first time in the last weeks of pregnancy or during the immediate postpartum period. These observations do not, however, apply with anything like the same force to acute pulmonary œdema (Fig. 1). This shows that about half our cases of pulmonary œdema occurred at the time of confinement or in the immediate post-partum period, and the others occurred at any time from the 14th to the 34th weeks of pregnancy though even this large series of pregnancies is scarcely sufficient to allow us to assess the relative distribution during the pregnancy. Fig. 1 also shows that pulmonary œdema occurred in 8 patients who were free from

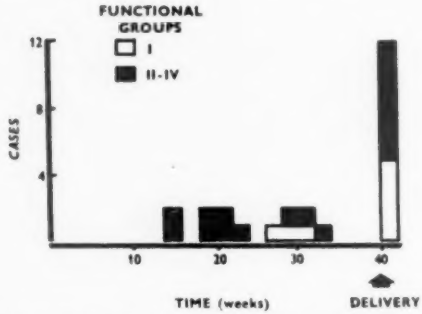

FIG. 1.—Pulmonary œdema in pregnancy. 25 cases. 8 deaths.

symptoms before pregnancy, and two of these patients died.

Mitral valvotomy and pregnancy.—Mitral valvotomy has introduced new possibilities in the management of pregnancy in patients with mitral stenosis. There are two ways in which it might be useful; first, in patients already pregnant, valvotomy might reduce the risk and, secondly, if performed before pregnancy, it might make pregnancy feasible, when it would otherwise be inadvisable.

Valvotomy during pregnancy: Valvotomy does not, of course, cure mitral stenosis, but it usually considerably reduces the valvular obstruction. The extent to which the stenosis can be relieved is variable, and in some cases the valve may not be much improved, though this is less common now than in the early days of mitral surgery. Several questions at once arise: (1) Is the risk of valvotomy increased if it is undertaken during pregnancy? If it is, then it might be wiser to wait until after pregnancy, whenever possible. (2) Is the risk to the child increased by operation during pregnancy? (3) Is the risk of pulmonary œdema associated with pregnancy materially reduced by operation?

The data at present available are not extensive but, including 12 of our own cases, 89 valvotomies during pregnancy have been recorded. In these cases there have been 2 maternal deaths, so it seems safe to say that the operative mortality is not increased, and that the risk of operating on cases of this kind may possibly be less than the combined risk of allowing the pregnancy to continue and operating subsequently. On the other hand, the mortality may well be higher than in the medical management of our cases of heart disease of moderate severity (1·1% in 343 cases). This group of cases is not, however, strictly comparable to the group in which valvotomy was undertaken.

The risk to the child does not seem to be increased; in 89 pregnancies 5 children were lost (5·6%). This compares favourably with the fœtal mortality of 8·6% in cases of heart disease cited by Hamilton (1947) and 10·2% in 990 pregnancies in 1946 to 1951 in my department. Thus there appears to be no valid objection to mitral valvotomy during pregnancy on the basis of risk to mother or child.

But does valvotomy reduce the risk of heart failure during pregnancy? In cases in which valvotomy is undertaken during pregnancy it is difficult to answer this question, for it is almost impossible to devise an adequate control series; it is possible, however, to get some information from cases in which pregnancy follows valvotomy.

Pregnancy following valvotomy: We have observed 16 pregnancies following valvotomy in 15 patients. None of these patients died in the post-valvotomy pregnancies but 7 pregnancies were not free from trouble, for 3 developed orthopnœa and nocturnal dyspnœa, and 2 others had frank pulmonary œdema, while one patient developed congestive heart failure, and one had frank hæmoptysis from pulmonary congestion. In this case and in one of the patients with nocturnal dyspnœa, the operation was apparently entirely satisfactory, but in 3 of the other cases the surgeon was unable to achieve a satisfactory final valve size at operation, and in the other 2 cases the heart disease was advanced with auricular fibrillation and much cardiac enlargement.

All these patients had a good result from operation in the sense that symptoms were relieved, yet at least one-third of them developed complications due to considerable pulmonary congestion in a subsequent pregnancy. This is a clinical illustration of the anatomical fact that mitral valvotomy reduces but does not cure mitral stenosis. Pregnancy, owing to the increased heart output and blood volume, exaggerates the symptoms of mitral stenosis, so that a degree of mitral stenosis which permits a tolerable, and even active life, may still lead to dangerous symptoms if the patient becomes pregnant. The best results are obtained only if a really good split is achieved, and if the surgeon is completely satisfied with the result trouble in a subsequent pregnancy is uncommon.

10 patients had also been pregnant before their valvotomies and there can be no doubt that they did better in their post-valvotomy pregnancies (Table II).

These observations should not be taken to indicate that valvotomy is frequently advisable in pregnancy. In my department we supervise

TABLE II.—PREGNANCY PERFORMANCE BEFORE AND AFTER VALVOTOMY (10 PATIENTS)

	Before	After
Number of pregnancies	15	10
Complications	8	3
Fœtal deaths	8*	4†
Maternal deaths	0	0

*2 non-cardiac.
†3 non-cardiac.

annually about 100 pregnancies in patients with heart disease yet, in several years, we have undertaken only about 15 valvotomies during pregnancy. But if the number of patients needing valvotomy during pregnancy is small, so is the number liable to develop pulmonary œdema. Our aim is to try to recognize as many as possible of these patients, so that valvotomy may be considered early in the pregnancy, while termination is still possible, if it seems preferable. It has become very clear to us that there exists a group of patients liable to develop pulmonary œdema during pregnancy although free from symptoms before pregnancy. These patients always have good signs of mitral stenosis, but the transverse diameter of the heart is rarely increased, and many are free from appreciable pulmonary congestion before, and often after pregnancy, yet rapidly develop congestion at some stage of pregnancy or during the puerperium. These patients need further study; all we can say at present is that in such cases the old criterion of the patient's capacity for effort before pregnancy is no measure of the risk of pulmonary œdema during pregnancy.

Congenital heart disease.—Owing to the comparative infrequency of congenital heart disease in pregnant women, and the wide variety of lesions that may occur, it is very difficult to collect an adequate series of cases, and the only condition which has been well studied during pregnancy is coarctation of the aorta. The present series is collected from nearly 2,000 supervised pregnancies in patients with heart disease and also by reviewing some of our other patients with congenital heart disease details of whose pregnancies have been recorded. There are 122 patients with the following lesions (Table III):

TABLE III

Atrial septal defect	36
Persistent ductus arteriosus	24
Coarctation of the aorta	14
Pulmonary stenosis	13 (3 with shunts)
Ventricular septal defect	11
Eisenmenger's syndrome	8 (1 death)
Aorto-pulmonary communication	3 (1 death)
Fallot's tetralogy	2
Other	11
	122

These 122 patients had 236 pregnancies with only two deaths. 12 others had severe symptoms and 11 mild or moderate symptoms. There were 186 living children, 28 spontaneous abortions (often in cyanosed patients), 16 therapeutic abortions and 6 still-births. Now that we know the overall results in this group, we consider that many of the 16 therapeutic abortions might have been avoided. It is not possible here to analyse the effects of pregnancy in most of these conditions but the results in coarctation of the aorta are interesting. In the past this has been regarded as almost the only cardiac indication for Cæsarean section, on the grounds that death was often due to vascular accidents and it was thought that labour might provoke this complication. Burwell and Metcalfe (1958) have, however, pointed out that most of the vascular accidents recorded have, in fact, not occurred during labour and they advocated normal delivery, as in other cardiac conditions. Our 14 patients had 33 pregnancies, there were no deaths and only one patient became breathless during pregnancy, which is almost abnormally normal! One patient had 9 pregnancies without trouble. Of the 33 pregnancies, 25 went to term, and only 8 Cæsarean sections were undertaken. We now believe that these were unnecessary.

In most forms of congenital heart disease pregnancy is remarkably safe. Trouble has, in our experience, arisen only in a few patients who were constantly cyanosed (and few of these patients have living children) or had considerable right ventricular enlargement due to pulmonary hypertension.

Acknowledgments.—I wish to acknowledge my indebtedness to many colleagues, especially Drs. S. Oleesky, E. G. Wade, J. Mackinnon, E. Ikonen, G. Howitt and D. E. Anderson.

REFERENCES

BURWELL, C. S., and METCALFE, J. (1958) Heart Disease and Pregnancy: Physiology and Management. London; p. 170.

HAMILTON, B. E. (1947) *Amer. Heart J.*, **33**, 663.

——, and THOMSON, K. J. (1941) The Heart in Pregnancy and the Child-bearing Age. Boston; p. 52.

Dr. F. Dudley Hart (London):

I intend to cover only those corners of the field of pregnant medicine in which I have first-hand experience.

Rheumatoid arthritis.—In the field of rheumatology, it is widely appreciated that patients with rheumatoid arthritis frequently go into spontaneous remission when they become pregnant. The usual course is for remission to

start within the first three months of cyesis. Joints become less swollen, there is less morning stiffness, less pain throughout the day, a lower sedimentation rate, and an all-round improvement in signs and symptoms. This remission is for the patient a blessing and a welcome release from pain, but it does not last long, for, when the baby has left her body, commonly within 2–3 weeks of its birth or even within the first few days, relapse sets in, doubly unpleasant after the previous holiday from arthritis and one difficult to tolerate, even without the new baby to care for. This is, however, by no means the general rule. The following two cases occurred within the last two years in my unit:

Case I.—Mrs. P., aged 33, became pregnant with her third child when she had been suffering from quite severe rheumatoid arthritis for two years, necessitating two hospital admissions and steroid therapy (triamcinolone) to control her symptoms. From the beginning of pregnancy, her arthritic symptoms became worse and they remained worse throughout pregnancy. Triamcinolone dosage had to be adjusted from time to time, but averaged 12–10 mg. throughout her pregnancy. Her condition further deteriorated about the 30th week and the fœtal heart was no longer heard. She was delivered of a macerated fœtus and thereafter slowly improved. It seemed that, from the very inception of pregnancy, her arthritis deteriorated and continued to do so as long as pregnancy continued. There was no immediate improvement when the child died, but a slow and gradual one after childbirth. It was quite the reverse of the usual picture: pregnancy here caused not remission, but relapse.

Case II.—Mrs. T., aged 34, had an established and fully compensated mitral stenosis, dating from rheumatic chorea at the age of 11 years. In 1952, at the age of 27, she developed rheumatoid arthritis. Two years later, as her condition was deteriorating, cortisone, 50 mg. a day, was started, but was discontinued within three weeks as, although there was improvement in her arthritic features, dyspepsia and dysmenorrhœa became severe. Later steroid therapy was recommenced, first corticotrophin, then prednisolone 10–15 mg. daily. Although dyspepsia remained a trouble, no ulcer was revealed in a barium series. In June 1957 she became pregnant and her pains improved, but she was unable to reduce her dose of prednisolone and fresh swellings appeared in the next few weeks and months. In February 1958 she was delivered of a macerated fœtus at the 35th week. Two months later, in April, her arthritis gradually relapsed.

In these 2 cases, the first relapsed from the start of her pregnancy and gradually remitted after childbirth; the second improved in some respects on becoming pregnant, though new lesions appeared while she was pregnant, and she only gradually relapsed after childbirth. Both had premature births and were delivered of a macerated fœtus. The question that is inevitably asked is, "Is there any relation between this fatality and the steroid therapy given in both cases?" Jones and Howard (1953) concluded that, given the medical indication, there was no obstetric contraindication to the use of ACTH or cortisone during pregnancy, but advised caution in the use of high dosage early or late in pregnancy, commenting on the possibility of causing serious depression of the fœtal adrenal glands. Guilbeau (1953) reported, in 30 patients treated with 50 mg. cortisone daily in the first trimester, one case of club foot, one coarctation of the aorta, and one premature (32 weeks) delivery of a child who died on the third day. DeCosta (1955) noted that, though cortisone may interfere with pregnancy in animals, there was no evidence of this in the human. He found, however, no good evidence that cortisone was beneficial in hyperemesis, toxæmia or erythroblastosis. Barnes (1954) warily states, "it is known to be safe to give cortisone during pregnancy and there is no evidence that it will produce fœtal damage. On the other hand, there is as yet, no absolute evidence that it will not produce fœtal damage. Probably the safest rule for the patient with intact adrenals is that, if at all possible, additional cortisone be withheld until the end of the third month, when genital differentiation of the fœtus is well completed". In an editorial in the *New England Medical Journal* in 1956, the writer noted that cleft palate without harelip had been produced in offspring of different strains of house mice, the incidence varying from 4 to 100, depending on the genotype of the mother and fœtus. The dose of cortisone and the period of gestation during which cortisone was begun were, he considered, important factors in addition to the genetic construction of the animals in determining the frequency of this defect. In rabbits, a high incidence of cleft palate without harelip could also be produced. The writer quotes the case of Harris and Ross (1956) where the defect occurred in a full-time stillborn child of a woman with idiopathic steatorrhœa treated with cortisone since the 8th week of pregnancy. Several such cases have been described from different countries, but care must be taken in drawing any conclusions as to cause and effect, for in the majority of cases reported normal children have been born of mothers on steroid therapy throughout pregnancy, even in such severe cases as systemic lupus erythematosus and acute and chronic leukæmia, in some instances the mother only surviving the birth of a normal child by a few weeks. Tenney and Little (1958) state, "with

respect to hormone therapy during pregnancy, it was stated that cortisone is no longer considered a panacea for all illness. In pregnancy, it is now known to be of little or no value in the treatment of toxæmia, pregnancy complications and Rhesus sensitized mothers. . . . It is now known that no effect on the human fœtus has been observed in patients who have received cortisone therapy throughout pregnancy in a dosage of over 300 mg. daily."

In view of our two fœtal fatalities, where the mother had conceived and been on steroids throughout pregnancy, I have asked a number of my colleagues for their experiences. Dr. Oswald Savage of the West London Hospital tells me of five normal confinements with normal children born, two on prednisolone, three on corticotrophin. Dr. George Kersley, of Bath, had nine pregnancies in rheumatoid arthritics, 7 improved and 2 deteriorated during pregnancy. He gave no steroids in any of these cases. Professor E. G. L. Bywaters tells me of 2 miscarriages, 1 on cortisone and 1 on prednisolone, and 1 case where an otherwise normal child was born with cleft palate. Dr. George Fearnley tells me of one patient of his who conceived on 7·5 mg. prednisolone daily, went into natural remission and discontinued steroid therapy in the 4th month—the full-time child born was entirely normal. Dr. A. J. Popert, by courtesy of Dr. W. S. C. Copeman, has kindly given me details of the 5 cases mentioned by Dr. Savage. One woman developed an increased response to corticotrophin during her first two months of pregnancy and developed features of Cushing's syndrome although dosage remained unchanged and, indeed, was gradually reduced subsequently. He also gives me details of a mild case of systemic lupus erythematosus who, taking 20 mg. of prednisolone when she became pregnant, was able to halve this dosage from the 16th week onwards. A normal child was delivered at full term and breast feeding was continued for several months.

On this rather patchy evidence, therefore, I can find only 5 cases in my own and my colleagues' series where fœtal fatalities occurred, with 2 instances of cleft palate on prolonged steroid therapy. If pregnancy occurs on steroid therapy in rheumatoid arthritis, natural remission usually allows one to reduce the dose of drug or even, very occasionally, as in Dr. Fearnley's case, to stop it altogether. One should continue on as low a dosage as maintains the patient's arthritis under reasonable control, in the full knowledge that the steroid will probably have to be increased again when symptoms exacerbate in the puerperium. But where pregnancy aggravates and worsens the rheumatoid condition such a course is not possible and steroid therapy may even have to be increased as pregnancy advances. There seems to be no evidence of risk of life to mother or fœtus in rheumatoid arthritis as such: on steroid-treated arthritis, however, the 5 fœtal deaths mentioned above make one a little more cautious.

Ankylosing spondylitis.—The reason we prefer to keep this disorder apart from rheumatoid arthritis is that the manifestations, progress, treatment and prognosis are so very different in the two conditions. Further differences are apparent when it comes to studying the effect of pregnancy and childbirth. Since it is more common in males than females, in a ratio probably around 5–6 to 1, ankylosing spondylitis is frequently overlooked in women. This disease in females frequently tends to be mild and is often largely or entirely confined to the sacroiliac joints for several years, and these are the early years when pregnancy is more likely to occur. We have been impressed by the fact that, unlike the state of affairs in rheumatoid arthritis, remission does not commonly occur during pregnancy, nor relapse in the puerperium. We have now had 14 female spondylitics who became pregnant and were delivered normally of 18 normal babies in all but one severe case, who had a Cæsarean section performed (Hart and Robinson, 1959). The child was normal. Steroids were not given in any of these cases. There was only one miscarriage in 19 pregnancies in these 14 spondylitics. There was no sign of the true remission often seen in rheumatoid arthritis. It is not uncommon, however, for the additional trauma of looking after a small baby to cause some symptomatic aggravation for a time in the puerperium, but true relapse is rare. The natural course of the disease appears to be little diverted by pregnancy or childbirth, and the stiff pelvis and back do not appear to cause undue obstetric difficulties. Our most severe case, with a completely fused pelvis, spine and hips, and a vital capacity reduced to 61% of normal, was the only one where a Cæsarean section was performed.

Diabetes.—There is a voluminous literature on the subject of pregnancy in diabetes mellitus. Although the diabetic condition may worsen and balance become slightly more difficult, the main risk is not to the mother, but to the fœtus. This has led to prolonged arguments as to the best way to reduce fœtal mortality. All agree on the value of close observation of the mother throughout pregnancy, and of the child after birth, and of extra careful diabetic control.

Most agree with termination of pregnancy around the 36th week, usually by Cæsarean section. Argument still abounds as to the value of hormones in the latter part of pregnancy, for, while the Medical Research Council's report (1955) found no difference in two groups of patients treated, one with large daily doses of ethisterone and stilbœstrol by mouth, and the other with inert substances, White *et al.* (1956) claim excellent results with stilbœstrol and progesterone given by intramuscular injection. Most of us feel that this prophylactic use of hormones remains non-proven and its rationale (Smith and Smith, 1935) open to doubt. As regards cortisone and its analogues, Dr. Wilfred Oakley tells me he is happy for his diabetic patients to receive this form of therapy, should rheumatoid arthritis develop. We have yet to see a diabetic rheumatoid arthritic woman conceive on cortisone.

REFERENCES

Barnes, A. C. (1954) *Obstet. Gynec., N.Y.*, **3,** 322.

DeCosta, E. J. (1955) *J. Ark. med. Soc.*, **51,** 180.

Guilbeau, A. A. (1953) *Amer. J. Obstet. Gynec.*, **65,** 227.

Harris, J. W. S., and Ross, I. P. (1956) *Lancet*, i, 1045.

Hart, F. D., and Robinson, K. C. (1959) *Ann. Rheum. Dis.*, **18,** 15.

Jones, G. S., and Howard, J. E. (1953) *N.Y. St. J. Med.*, **53,** 2463.

Medical Research Council Report (1955) *Lancet*, ii, 833.

New Engl. J. Med. (1956) **255,** 623.

Popert, A. J. (1959) Personal communication.

Smith, G. V., and Smith, O. W. (1935) *Surg. Gynec. Obstet.*, **61,** 175.

Tenney, B., and Little, A. B. (1958) *New Engl. J. Med.*, **259,** 625.

White, P., Gillespie, L., and Sexton, L. (1956) *Amer. J. Obstet. Gynec.*, **71,** 57.

Mr. Charles M. Flood (London):

It is probable that every known medical disease has afflicted some pregnant woman, somewhere at sometime. I shall deal with only a few of the diseases which, in this country at any rate, are of concern to the obstetrician, because of their frequency and because of their effects both on mother and on child, if they be not diagnosed and effectively treated.

Pulmonary tuberculosis, asthma and emphysema, cardiovascular disease, hypertension and anæmia, diabetes, thyrotoxicosis, chronic nephritis, and disease of the central nervous system, together with psychiatric disease, form the vast majority of the medical diseases complicating pregnancy. On psychiatric disease in pregnancy, I would merely remind you that, at a meeting of the Section of Psychiatry on February 12, 1957, although figures were reported from University College Hospital showing that 40% of some 500 terminations there in eighteen years had been for psychiatric reasons, the meeting generally failed to agree on the criteria for advising the termination of a pregnancy for psychiatric reasons (*Proceedings*, **50,** 321). Many speakers stressed that the treatment of the pregnant and non-pregnant psychotic differed not at all, except that the roles of the social and moral welfare workers were even more important in the pregnant patient. Termination never solved one set of psychiatric problems and very often raised new ones.

Physiological changes occurring in normal pregnancy.—The total blood volume increases by up to 25% (Dieckmann and Wegner, 1934) and the uterine vessels at term contain approximately one-sixth of the total maternal blood volume. The total number of red corpuscles and the total amount of hæmoglobin in the body increase, though to a lesser extent than the total plasma volume, resulting in the so-called "physiological anæmia of pregnancy" (Strauss, 1934), though recent work has cast doubt on this long accepted concept (Camilleri, 1958). Cardiac catheterization shows that the output rises markedly at the third month, to reach a level at least 30% above normal by mid-pregnancy, falling suddenly after the thirty-sixth week to reach levels almost normal just before term (Palmer and Walker, 1949; Hamilton, 1949). However, dilatation of the vascular bed, chiefly due to the utero-placental shunt, coupled with the decreased viscosity of the blood, prevents any rise in blood pressure in normal pregnancy. It is probable, though not certain, that the heart hypertrophies in pregnancy. The lower thorax is lifted and expanded in late pregnancy. The diaphragm is raised, but the dome remains flat, the uterine fundus lying not under the dome but under the subcostal arch. The vital capacity is normal or increased as is the pulmonary ventilation (Thomson and Cohen, 1938). The metabolic rate rises from 5 to 35%. Carbohydrate storage and mobilization are slower than in the non-pregnant state. Alimentary glycosuria is not uncommon in early pregnancy though rare later. So long as an adequate diet ensures 1·5 grams of calcium daily there is no negative calcium balance, though there may frequently be so during lactation. The fœtus, uterus, placenta and blood loss at delivery correspond to about 725 mg. of iron with a further 180 mg. for six months' lactation, calling for a minimum absorption of 3 mg. of iron daily throughout pregnancy. The chronic iron deficiency common even in non-pregnant women is further aggra-

vated by diets deficient in iron as well as by the hypochlorhydria often occurring in pregnancy. Vitamin deficiency in Britain is as rare in pregnant women as it is in the non-pregnant, so that the routine distribution of vitamins is a waste.

Pulmonary tuberculosis.—The work of Cohen (1946) and of Stewart and Simmonds (1947), together with the great advances in the last few years in the medical and surgical treatment of pulmonary tuberculosis, has radically altered former views on tuberculosis in pregnancy.

TABLE I.—BLACK NOTLEY MATERNITY UNIT (*July* 1937–*September* 1948)

Type of case	Number of cases	Number showing retrogression
Arrested and recovered	107	4
Quiescent	83	8
Active	89	20
Totals	279	32 (11·5%)

From his results (Table I) Cohen concluded that the proportion of retrogressions was no more than would have been expected in a comparable group of non-pregnant women under similar conditions. Of the 32 cases who retrogressed, 11 improved or became quiescent before discharge and of the 20 active cases who retrogressed, 12 were hopelessly advanced when they first came under treatment. The late follow-up results were comparable and as favourable. Pregnancy is an incident, albeit an important one, in the natural course of the disease. Relapses after pregnancy are more often due to social and economic factors rather than to the direct effects of the pregnancy. I do not advocate wholesale fecundity in the phthisical but merely a more enlightened attitude than that shown by the classical dictum: For the virgin—no marriage; for the married—no pregnancy; for the pregnant—no confinement; for the mother—no suckling.

Heart disease.—Mackenzie (1921) was the first to insist that the functional ability of the heart was of more importance than the structural lesions therein and to him pregnant women owe a great debt. The earlier in pregnancy that the patient can be seen and assessed the better, not only because of instituting early and adequate treatment, but also because the normal physiological changes of pregnancy may simulate cardiac disease. Dyspnœa, tachycardia, œdema, basal crepitations, distension of the cervical veins, altered heart sounds and added murmurs, arrhythmias and fainting attacks, all these may equally well be present in normal pregnancy as in cardiac disease. Cyanosis, hepatic enlargement, or hæmoptysis, however, do not occur in normal pregnancy. Antenatal care, is directed to preventing the cardiac deterioration of the woman and at its best can achieve magnificent results. O'Driscoll *et al.* (1957) of Dublin report a series of 539 such pregnancies with six maternal deaths attributable to pregnancy giving a maternal mortality of 1·1% in a series, rare for its high age and parity, in a city where contraception, termination and sterilization are not practised. Similar results are reported from Edinburgh in the twenty years from 1928 to 1947 with progressive improvement in the maternal mortality from 6·3% to 0·9% (Gilchrist and Haig, 1950). Such results confirm the opinion of Gilchrist and Murray-Lyon (1933) that pregnancy, if properly supervised, does not affect the natural course of heart disease. Here I wish to make three points:

(1) As the burden on the heart decreases after the 36th week, rest is often of more value before this time than after it.

(2) Though most cardiac patients have swift, easy labours, and long, exhausting labours are to be avoided, the mortality after Cæsarean section is at least twice that after vaginal delivery.

(3) The few hours after delivery are most critical, for the closure of the utero-placental shunt may result in acute pulmonary œdema when venesection and intravenous morphia will be life-saving. Though it is possible that the swift uterine contraction, following routine intravenous ergometrine favoured by some obstetricians, may increase the circulatory blood volume precipitately, I do not favour allowing cardiac patients to lose blood through a "routine" postpartum hæmorrhage. This can often be excessive and uncontrollable whereas a venesection is always under immediate control.

Hypertension in the non-pregnant is unpredictable enough, but hypertension in the pregnant is even more so, as can be seen by briefly reviewing 6 diverse cases:

I.—A primigravida, with essential hypertension, who, within eight weeks of delivery, died of malignant hypertension.

II.—A primigravida with moderate hypertension, who, within two years of delivery, was dead from a cerebral hæmorrhage.

III.—Four pregnancies in a woman who had no previous hypertension, but who developed essential hypertension after the third pregnancy.

IV.—A woman with essential hypertension. Her five pregnancies were complicated by severe exacerbations but her hypertension has not deteriorated over the years.

I am indebted for these details to Mr. G. F. Gibberd under whose care these 4 patients were.

V.—A patient with mild hypertension who became

progressively a little worse after each pregnancy until finally, at the age of 37 years, her blood pressure is 200/130. It is reasonable to attribute this deterioration to each successive pregnancy.

VI.—The blood pressure in her identical twin sister who has never been pregnant is virtually the same. Platt (1958) reported this history and he noted that the brother and mother of the twin also had hypertension and are now both dead.

Thus when the immediate, let alone the remote, fate is totally unpredictable, the best we can do is to wait and see what happens. Too harsh an interdict in Case IV for example would have deprived the patient of much happiness—including 8 grandchildren!

Anæmia.—The treatment of anæmia in pregnancy is usually the responsibility of the obstetrician, but the complacency of many in this respect is appalling. No general surgeon would embark on routine major surgery with the patient's hæmoglobin at a level considered adequate by many obstetricians for the unforeseeable hazards of labour. From 5 to 10% of labours end with a postpartum hæmorrhage of more than a pint. Since the majority of anæmias in pregnancy are due to iron deficiency, it is easy to be lulled into a sense of false security by the administration of iron, especially if given routinely to all antenatal patients, unless frequent checks are made to see that the hæmoglobin level *is* in fact rising. If it does not rise, then fuller investigation is called for, including if need be sternal puncture and gastric analysis. Intravenous or intramuscular iron may be of value, but there should be no hesitation in transfusing patients with severe anæmia near to term. The risks of blood transfusion are slight indeed compared with the results of anæmia in pregnancy, especially an increased incidence of inertia, hæmorrhage, sepsis, thrombosis and pulmonary embolism.

Diabetes.—On the other hand, to guide the pregnant diabetic through her pregnancy is almost entirely the task of the physician, the obstetrician being left to decide how best to terminate the pregnancy, speedily, safely and usually at about the 36th week. The value of strict control and treatment is shown by the reduction in maternal mortality from 45% fifty years ago (Williams, 1909), to 0·4% reached by Joslin in 1946. The fœtal loss has fallen, though less dramatically, by about half to some 25%, except in a series by White (1947) in which, by the use of œstrogen and progesterone, she claims a fœtal loss of only 10%. It is of interest, too, that parous women who develop diabetes in middle life may give obstetric histories very similar to those of the long-established diabetic. In this pre-diabetic state, Peel and Oakley (1950) found a fœtal loss of 23% and 27% of the babies weighed more than 10 lb. (4·5 kg.) in 237 such pregnancies. It would seem that the diabetogenic and growth factors of the anterior pituitary are closely linked.

Two points should be emphasized: (1) The pregnant diabetic should be under the close care of the physician who has been supervising her diabetes when she was not pregnant. (2) The finding of a reducing substance in a pregnant woman's urine means that she has diabetes until the most thorough tests have proved the contrary.

The birth of a child is of importance, not only to a woman, often being the fulfilment of her fundamental desires and greatest hopes, but also to her husband and to her children, especially to what might else be an only child. It is thus not lightly to be prohibited by medical men who dictate when they should only advise. Also, it should always be remembered that it is the pathological that complicates the normal. The true outlook, and one that I should like to see more often, is, not that normal pregnancy is complicating a pathological process, but that it is the abnormal disease which is complicating an otherwise normal pregnancy.

REFERENCES

CAMILLERI, A. P. (1958) *J. Obstet. Gynæc., Brit. Emp.*, **65**, 266.

COHEN, R. C. (1946) *Brit. J. Tuberc.*, **40**, 10.

DIECKMAN, W. J., and WEGNER, C. R. (1934) *Arch. intern. Med.*, **53**, 527.

GILCHRIST, A. R., and HAIG, D. C. (1950) In: Modern Trends in Obstetrics and Gynæcology. Edited by K. Bowes. London; p. 327.

——, and MURRAY-LYON, R. M. (1933) *Edinb. med. J.*, **40**, 587.

HAMILTON, H. F. H. (1949) *J. Obstet. Gynæc., Brit. Emp.*, **56**, 548.

JOSLIN, E. P. (1946) Treatment of Diabetes Mellitus. London and Philadelphia.

MACKENZIE, J. (1921) Heart Disease and Pregnancy. London.

O'DRISCOLL, M. K., BARRY, A. P., and DRURY, M. I. (1957) *Brit. med. J.*, ii, 1090.

PALMER, A. J., and WALKER, A. H. C. (1949) *J. Obstet. Gynæc., Brit. Emp.*, **56**, 537.

PEEL, J. H., and OAKLEY, W. G. (1950) Transactions of the 12th British Congress of Obstetrics and Gynæcology, 1949. London; p. 169.

PLATT, R. (1958) *J. Obstet. Gynæc., Brit. Emp.*, **65**, 385.

STEWART, C. J., and SIMMONDS, F. A. H. (1947) *Brit. med. J.*, ii, 726.

STRAUSS, M. B. (1934) *J. Amer. med. Ass.*, **102**, 281.

THOMSON, K. J., and COHEN, E. M. (1938) *Surg. Gynec. Obstet.*, **66**, 591.

WHITE, P. (1947) *Penn. med. J.*, **50**, 705.

WILLIAMS, J. W. (1909) *Amer. J. med. Sci.*, **137**, 1.

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JOINT MEETING NO. 4

Section of Odontology with Section of the History of Medicine

Chairman—W. G. SENIOR, C.B.E., Ph.D., F.D.S.
(President of the Section of Odontology)

Meeting
April 27, 1959

C. E. WALLIS MEMORIAL LECTURE

Methods and Materials Used for Artificial Teeth [*Abridged*]

By R. A. COHEN, L.D.S., F.S.A.
Warwick

My first duty is to speak of Charles Edward Wallis, whose name is commemorated in this lecture. He was born in London in 1869, and received his professional education at King's College Hospital and the Royal Dental Hospital. He joined the staff at King's as Assistant Dental Surgeon in 1899 and became interested in the dental treatment of school children. He was elected Dental Surgeon in 1911 when A. Swayne Underwood retired. It was largely due to his writings that the first London County Council School Dental Clinic was established and he was appointed to supervise these clinics. He was active in the affairs of the British Dental Association, and widely read in history and archæology. He held several honorary appointments and published a number of papers on school dental clinics and historical subjects. He died suddenly in 1927.

I am the first Wallis Lecturer to be sufficiently junior not to have had the privilege of knowing Wallis personally, and I am indebted and grateful to Mr. S. E. Wallis for these facts, which are taken from his unpublished book on the history of King's College Hospital Dental School.

The literature on artificial teeth is unexpectedly large, but unfortunately the period before Fauchard, while abounding in passing references to artificial teeth, provides very few details of their material or construction. The eighteenth and nineteenth centuries, however, which saw great development in the art, are more fruitful, since a number of textbooks for the instruction of dentists were published, besides a flood of advertising material. I have thought it best to confine my attention to original works I have myself examined, with a few exceptions, and to examples of dentures I have actually handled. Although it has been impossible to cover the whole field or to visit every museum where old dentures are to be found, it is hoped the additional accuracy so obtained will compensate for the necessarily restricted survey.

I propose to deal with removable artificial teeth, except in the case of Etruscan dentures, and not to touch on crowns or the history of porcelain teeth, and to end with the introduction of vulcanite in the middle of the nineteenth century.

A very few examples of prostheses have been discovered, the earliest dating from 2500 B.C., having their origin in Egypt, Phœnicia and Greece, but according to the physical evidence now available it must be agreed that the Etruscans were the first people to make artificial teeth in any numbers.

The Etruscans were a people who inhabited at one time the greater part of Italy before the founding of Rome, perhaps between the ninth and third centuries B.C. They were skilled metal workers and architects and many examples of their buildings are still in existence. From the large number of graves excavated have been obtained numerous interesting everyday objects and beautiful specimens of jewellery and metal work including artificial dentures. As may be expected the majority of such dentures are in Italian museums but there are two examples in this country, both in the Liverpool Museum (Fig. 1). They are in a collection formed by Joseph Mayer and presented to the city by him in 1867. Nothing is known of the history of the objects before they came into the possession of Mr. Mayer. The collection is particularly rich in gold jewellery, nearly all in extremely good condition, and is dated from the seventh century B.C.

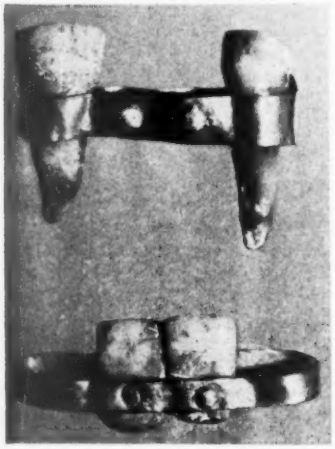

FIG. 1.—Etruscan dentures. Upper specimen: central incisors (? not original) as abutments, artificial teeth missing. Lower specimen: abutments missing, artificial teeth (human) present. (By permission of the Liverpool Libraries, Museums and Arts Committee.)

to the third or second century B.C. when gold working as an art died out. The collection has been described in 1932 by Mary A. Johnstone [1], and in at least one Italian publication. The two dentures are illustrated by Johnstone and a very accurate description given, but as far as I can discover, have not been illustrated in any dental history. The earliest reference I can find to the Liverpool specimens is a note in the *Independent Practitioner* (1885) by W. H. Waite describing the dentures [2].

From a study of illustrations of Etruscan artificial teeth it would appear that models of the mouth would be necessary for their construction although Johnstone makes the interesting suggestion that "it is possible that some, but certainly not all, of the gold dressings were added when the body was entombed in order to make it more comely."

It is likely that the practice of dentistry spread from Etruria to Latium, and from the numerous and well-known references in Martial and other Roman authors to artificial teeth, it appears to be certain that they were commonplace in ancient Rome, but not enough examples have survived to enable accurate evaluations to be made.

I have not been able to find any references to artificial teeth during the so-called Dark Ages, and it is not until the time of Albucasis (936–1013) that a reference can be found. This writer, after describing how loose teeth may be bound together and supported by gold or silver wire, states "Sometimes, when one or two teeth have fallen out, they are replaced in the sockets, bound in the aforesaid manner and they will remain there. . . . The vacancy left by fallen teeth can be filled with artificial ones, made of ox bone, they also being fixed in the manner above described; they will be found not only an advantage from the æsthetic, but also the functional point of view" [3].

The first illustration of artificial teeth is to be found in the works of Ambroïse Paré [4]. The following description is taken from the second English edition of the collected works translated by Thomas Johnson, 1649 [5]. ". . . other teeth artificially made of bone or Ivorie may bee put in the place of tho∫e that are wanting, and they mu∫t bee joined one fa∫t unto another, and al∫o fa∫tened unto the natural teeth adjoining, that are whole; and this mu∫t chiefly bee don with a thred of gold or ∫ilver, or for want of either, with a common thred of ∫ilk or flax, as it is declared at large by Hippocrates, and al∫o de∫cribed in this figure following." The original French also states that artificial teeth may be made of the *dents de Rohart* which Lindsay [6] states is from the Icelandic *rosmhvair* meaning a walrus.

Guillemeau, whose work on surgery was translated into English in 1597 [7] describes a paste consisting of white wax, gum elemi, white coral and "prepared pearls" which may be used for forming artificial teeth or filling cavities in teeth, the actual passage being "Farthermore anye bodye by arte may make teeth of white waxe which beinge meltede, & liquefactede with as much gumme Elemni, or a little ma∫tick, white Coralle, and preparede pearles & thereof a pa∫te beinge made, of the which we may forme as manye teethe as we plea∫e. This pa∫te is al∫o verye commodiou∫e to repleni∫h therewith a hollowe toothe, becau∫e ther might noe viandes remayne therin, through which the teethe doe more corrupte, and more intollerable payne is heer bye ∫u∫citatede [*sic*]". This has already been noted by Lindsay [6, 8]. Weinberger does not regard the paste of Guillemeau as of any value in the filling of the teeth saying "a tooth made from it could hardly have withstood the pressure of mastication and at its best could not have been more than a temporary filling" [3]. I have constructed some dentures using the paste and on the contrary, the material is exceedingly hard. For "prepared pearls" I have used mother-of-pearl, since it is hardly likely that pearls would have been ground up. It appears, therefore, that Lindsay was quite correct in regarding the paste "as a great advance in dental surgery." James Cooke (1676) says, "some make them [artificial teeth] thus, Cer. alb. Gum Elim. an

part. equal. Or, with Mastich, a little white coral prepared and made into a paste, of which Teeth may be framed: it may also be useful to stop Teeth that are hollow, to keep the Air out" [9]. Although no doubt Cooke was copying from Guillemeau, this passage does indicate that the paste was known to surgeons in the seventeenth century.

Peter Lowe (1654) states, regarding the treatment of loss of teeth, "for remedie whereof we make artificiall teeth of Ivory, Whales bone or hounds teeth, which ſhall be faſtened by a wyre or thread of gold, paſſing the wyre or thread betwixt the whol tooth on either ſide next adjacent, then put the artificiall tooth in the part, then knit the thread faſt through about the ends of the thread . . . I am not mindfull to inſiſt in this practick as I might, becauſe it is ſeldome practiſed" [10].

Nuck (1696) gives an interesting short account of artificial teeth, in which he says that nothing is more common than to make artificial teeth out of elephant ivory and having made them to tie them to the neighbouring teeth with a thread of gold or silk. But since they become stained by food or saliva in a short time they are better made from hippopotamus teeth, especially if the outer surface is of good colour. If all the teeth in the lower jaw fall out a complete set of teeth should be made from elephant ivory or hippopotamus teeth and so placed within the lip that with the movement of the jaw it also is moved and food taken into the mouth can thus be chewed [11].

The first detailed instructions for the making of artificial teeth are to be found in the wonderful textbook by Pierre Fauchard (1728). This book has now become rare and valuable, but fortunately there is available for English-speaking students the translation made by Lindsay and published by the British Dental Association in 1946. There is no doubt that this is one of the most considerable pieces of scholarship ever completed by a single dental historian, and all workers in this field must remain permanently in the translator's debt.

Fauchard says that human teeth are usually used and that the pulp cavity should be filled with lead, but that hippopotamus (or sea-horse) teeth, the teeth of oxen, and even the bones of their legs, the tusks of walrus and the core of ivory which is finest and most beautiful are also used. He must mean that teeth may be carved from hippopotamus tusk, ox bone, walrus tusk and elephant tusk. Ox teeth would appear to be too large, but calves' teeth might be suitable.

He goes into considerable detail about the drilling of the holes for linen thread, waxed silk or gold wire which served to retain the piece by being tied round the neighbouring teeth. Where a number of teeth are to be replaced, the row of artificial teeth is to be supported by a narrow band of gold, each tooth being fixed to it by a rivet which passes from the lingual to the labial surface. Sometimes an artificial piece can be supported by a post crown, which is cemented in place by the following: Flake gum lac two ounces, finest Venice turpentine half an ounce, white coral in powder very fine, two ounces. The gum is melted, the turpentine and coral added. The mixture is formed into little sticks and pulverised when required.

A little of the powder is put up the enlarged root canal and the post is warmed.

He also gives instructions how to prepare ox bones for artificial teeth. I have not been able to obtain ox bones, oxen being large draught animals, but I have prepared some leg bones of a cow more or less according to his instructions and it is apparent that no large denture could be made from such a material. Fauchard says he prefers ox bone to elephant ivory because the latter soon becomes yellow.

When a complete upper denture is to be made it is supported by a sort of frame fitted over and around the lower teeth, to which the upper plate is attached by flat steel springs which are inserted into the distal aspect of the last tooth and retained by threads through both the denture and the spring. He says "This machine combines not only the qualities of those which have preceded it, without their discomfort, but it has several other advantages which distinguish it and render it a hundred times more convenient. . . . The experts in this art in the attempts they have made at an upper denture have only used, up till now, springs of whalebone which are fastened to the natural teeth of the lower jaw with thread." Later he suggests that whalebone may be added to the steel springs he used to make them last longer. From this passage it is obvious that attempts had been made by dental practitioners in the seventeenth century to construct full upper dentures, although descriptions have not survived, and Fauchard states in a later passage that springs coiled like corkscrews or spirals were used before this time, but were not satisfactory. When complete upper and lower dentures are constructed the steel or whalebone flat springs are fitted into slots and tied into the denture; the springs are then covered with several layers of thread. Finally Fauchard describes a method of making a strip of gold or silver enamelled with colour to represent the gum and the tooth. The work is to be done by those who are used to working with enamel, and the strip is fastened to the bone base with rivets [12]. It is an extra-

ordinary thing that Fauchard does not mention the taking of impressions and, as Lindsay says, "it is certain that Fauchard knew nothing about impression taking, since there is no mention of such a thing in his work, and it is not likely that such an accurate and careful describer would have omitted to speak of this important point. He evidently measured the gums and spaces between the standing teeth with compasses, since he frequently speaks of taking the measure of the mouth" [8].

From the statements made by Fauchard it appears that he did not fix artificial teeth, whether human or animal, by means of posts in the pulp chamber, but that the rivets were at right angles to the long axis of the tooth. When dentures are seen with teeth fastened in this manner, they may well be assumed, therefore, to date from the first half of the eighteenth century, but I have yet to examine any, and it is thought that no teeth attributable to Fauchard now exist.

The first mention of a model is in a work by Purmann (1684). Lindsay gives a translation of the relevant passage, and while the meaning is not quite clear, it would appear to indicate that a model in wax is made in the mouth and a denture in bone or ivory constructed from it [8].

Pfaff (1756), however, specifically mentions taking an impression in sealing wax of edentulous jaws in two halves and from this a model is made. The sealing wax used would be largely beeswax and softened at a much lower temperature than the sealing wax now used [8].

Bourdet's book of 1757 is an important work, and the chapter and plate dealing with artificial teeth are in some respects superior to Fauchard's work, with which Bourdet was obviously well acquainted.

He says that artificial teeth are rarely made of ivory, i.e. elephant ivory, and that sea-horse is the best material because of its colour and solidity. He has often mounted a human tooth on a small piece of sea-horse, the tooth being retained by a rivet. When the gums have shrunk a long way, an artificial tooth may be made with enamelled gums and it is necessary to take exactly all the necessary measurements and to form a model in wax. Such dentures are retained in the mouth by threads, and need great care. Bourdet describes dentures for the four upper incisors which were retained by threads, the teeth being attached by two pins, one vertical and one transverse, and must have been very secure. Fauchard apparently did not use vertical pins. A number of natural teeth can be mounted on a piece of gold fitted to the gum, the pins and teeth are fitted, then removed and the piece sent to the enameller for colouring the gums. When it is enamelled, the teeth are mounted and cemented with Fauchard's mastic cement. Bourdet says that Fauchard only used the cement for crowns, but that he, Bourdet, used it to cement artificial teeth on to plates. The springs used by Bourdet appear to be flat, and he says that gold is better than whalebone or steel, and while he does not use whalebone, he sometimes places whalebone springs on the dentures temporarily to enable the patient to get used to them [13].

Berdmore (1768) discusses artificial teeth in general terms, and says that while gold ligatures often cut the teeth, this does not happen if silk twist is used. He says that complete dentures may be made for both jaws by the help of springs of a new and peculiar description, but unfortunately he does not describe them, merely saying "they are totally different in shape and action from those which have been used by my predecessors" [14].

I have a letter written by Dr. Erasmus Darwin dated March 18, 1785, to a Mr. Richard Dixon of Felsted, Essex, in which he states "If you could get false teeth, you would find that another consolation, as you would speak easier, and if you could get it (for it is but one piece cut to look like 2 or 3 teeth) made of ivory instead of the bone [bone deleted and horn substituted] of the sea-horse, it would become dusky and look like your other teeth. I should recommend Beardmore [*sic*] to you in Bolt-Court, Fleet Street. I advised my brother at Elston to get an artificial tooth, but I believe he thought it a sin and would not at all listen to me about it."

An interesting sidelight on the practice of dentistry in the late eighteenth century is seen in an unpublished letter in my possession, written on March 6, 1782, by Manette Talma who at that time was living with her father, the well-known dentist in London, to her brother, the future actor, in Paris. Manette says [trans.] "Father asks you to do everything you can to get him some teeth, and if you can go into the mortuary, to take advantage of the opportunity. If that is not possible, you must try and get to know the brother who is in charge of the place and ask him to let you have some; you will pay for them, the big incisors and the little laterals. Father used to pay his predecessor 12 livres a hundred for the canines. Don't forget all my messages. And the little molars [buy them] if they are fine and white, but much cheaper than the others." A livre was an alternative name for a franc and had the approximate purchasing power of the contemporary English shilling. Talma junior was apparently somewhat dilatory about executing his commission since M.

Georges Dagen of Paris has given me a copy of a letter written by Talma senior with a note also written by Manette and dated May 31, 1782 [trans.]. "You will not forget the teeth that Father asked you for, for he has not any more and needs them very badly."

There is a large number of patents dealing with the construction of artificial teeth from 1791, the earliest being that of Dubois de Chémant. Unfortunately the works of de Chémant from 1797 give no details of the manufacture of his mineral paste, but there is one statement which might be of value in deciding whether a particular denture is in fact of the period of de Chémant. He quotes from the "Report of the Academy of Sciences concerning the Teeth and Sets of Teeth of the new composition of M. Dubois de Chémant, Extracted from the Registers of the Royal Academy of Sciences 10 June 1789", where it is stated that "M. Brisson . . . found that a cubical inch of it weighed one ounce, two gros ($\frac{1}{4}$ oz.) and sixty nine pennyweights, whereas the lightest china of Seve [*sic*], of the seventeen kinds which he tried, weighs one ounce, three gros ($\frac{3}{8}$ oz.) and nine grains." The report is signed by D'Arcet and Sabatier [15]. It is not known to me how this weight would compare with the porcelain blocks and continuous gumwork of a later period, but it is a matter which would appear to be worthy of further investigation, when a full history of porcelain teeth is undertaken.

De Chémant's English patent dated May 11, 1791, gives some details of the process and the formulæ of the pastes. It is apparent that frequently three bakings were necessary. The procedure for constructing the denture was as follows: A quantity of soft wax was placed in the mouth, which was then shut giving an exact impression of the space required to be filled. In this was poured a composition formed of plaster of Paris, which when dry gave a true and solid model of the mouth. The well-kneaded paste was pressed into the plaster mould, removed, allowed to dry and then fired. As the paste in drying (and baking) lost some of its thickness it was necessary to spread or widen the wax mould when taken out of the mouth to an increase of about one-seventh, by pressing on the middle of it with the finger and thumb. Holes for the fastenings, that is threads or gold wires, had to be bored before the paste was quite dry. When the paste was dry enough to be handled, the teeth were carved in it with a sharp instrument before the piece was fired. After firing, the enamel colouring was painted on the teeth with a brush and the piece baked. Finally the gums were painted and the denture again fired (Fig. 2).

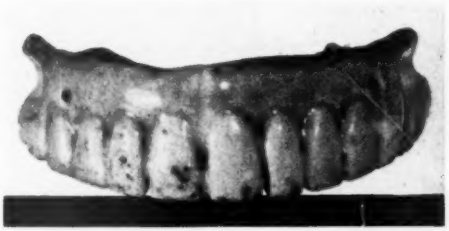

FIG. 2.—Upper denture, porcelain, believed to be by de Chémant, *c.* 1800. (The Odontological Museum of the Royal College of Surgeons.)

The last part of the patent specification concerns the making of spiral springs formed by twisting gold wire round a mandril.

Tomes' patent of March 3, 1845, is of considerable interest. It is in two parts, the first consisting of a method of obtaining a model of the proposed denture and the second the copying of this in hippopotamus or walrus ivory. A wax impression of the mouth was first taken and a plaster model made of the denture. The composition consisted of shellac, ivory dust or plaster of Paris and a solution of india rubber. This composition model was tried in the mouth and adjusted as necessary. It was then fixed in the machine and copied, the essential principle being the movement of a blunt point over the model transferred to a sharp point cutting the ivory. Reverse surfaces and undercut areas could be obtained. Tomes did not claim priority regarding the copying principle, there being in fact a number of such devices previously described, but he did claim that he was the first to use it for dental purposes. Unfortunately there is no example now in existence and there is no model of it in the Science Museum at Kensington, although they have a number of copying machines.

Of the remaining patents, comparatively few are of importance, and it is to be assumed that the majority of such patents were enrolled because of their advertising value.

Harrington patented a press for forming tortoiseshell into dentures in 1849 and an example of such a denture (Fig. 3) is shown by the courtesy of the Director of the Fauchard Museum, Paris, but it is not known if it was made by Harrington. I have tried to press tortoiseshell into dentures, but have not been able to give the matter sufficient time to have much success.

Laurie patented the process of John Allen for continuous gum work in 1853, using a platinum base, and Massey (1854) and Loomis (1854) also

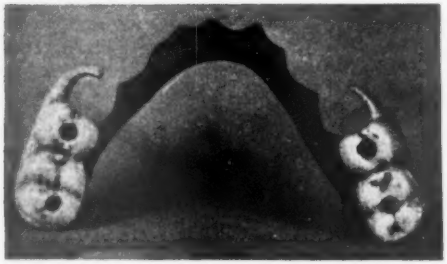

FIG. 3.—Lower denture, tortoiseshell and hippopotamus ivory, posts of silver or platinum and amalgam (?), *c.* 1850. (The Fauchard Museum, Paris.)

worked in porcelain. Charles Goodyear's famous vulcanite patent (the English patent) is dated March 14, 1855, but it is known he was experimenting for many years previously.

Rubinstein's patent of 1859 is of interest since it mentions the use of mother-of-pearl as a base.

Dental patents are a large and difficult subject, involving questions of priority between English, American and Continental patents and also priority between different workers, but it is a field which is worthy of the attention of dental historians.

Gariot, whose book was published in 1805, makes some interesting statements. He says that human teeth are very seldom used (i.e. in 1805), those of the sea-horse and those manufactured from mineral paste having been substituted for them. This is certainly not true of English dental practice of the period. He says that he has purchased from de Chémant the recipe for making his composition teeth, but he is not allowed to give it. He describes ligatures for retaining dentures in position and states that while springs for dentures may be steel or gold bands or whalebone, spiral gold springs are best [16].

The best account of dental practice regarding artificial teeth in the first quarter of the nineteenth century is to be found in the work of Maury (1828). There exists a picture of his workroom, reproduced by Dagen in 1926, but I have not been able to find an original example suitable for copying [17]. However, through the courtesy of Dr. Ernest Weil I am able to show a copy of a lithograph of Fattet's dental laboratory, dating from about 1840 (Fig. 4).

Maury states that materials used for artificial teeth have been the bones and teeth of oxen, horse, sheep, stag and several other animals, ivory, mother-of-pearl, teeth of the hippopotamus or sea-horse, and teeth made from mineral

FIG. 4.—From "La Vie de l'Illustrissime Inventeur des Dents Osanores" (Georges Fattet), *c.* 1840, Paris.

paste.[1] Persons who have lost their front teeth, he says, have for a long time replaced them with teeth made of white wax. He describes all these materials, giving their advantages and disadvantages, and states that at the present time (1828) hippopotamus teeth are much used, both with and without their enamel. Human teeth are far the best imitation and should be obtained from those who die in hospital, aged 18-40, and are brought to the amphitheatre for dissection. They should be carefully removed, cleaned and kept in bran, sand, fine grain or sawdust. Those from cemeteries are generally not suitable, as they become yellow and brittle.

Two models are necessary, one to fit the plate roughly, the other to finish on. The teeth adjoining the space to be filled should be scraped a little so that the piece may be a little larger than actually necessary. The cast is covered with black or red paint and the piece gradually cut with the rasp and the flat graver. Various types of teeth and dentures are described, and he states that the inner skin of the birch tree dipped in varnish is the best method of cementing human

[1]There is a good deal of confusion in the use of the names sea-horse, sea-cow and morse, the Oxford Dictionary giving all three names as synonyms for both walrus and hippopotamus. I believe that morse should be used for the walrus, sea-cow for walrus and sea-horse for hippopotamus. Ash's catalogues of 1851–1875 quote "Hippopotamus or Sea-horse teeth" and "Walrus or Sea-cow Tusks".

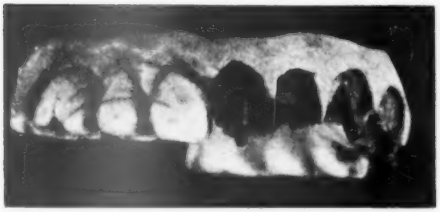

FIG. 5.—Upper denture, hippopotamus ivory, incisors (probably human) missing. Five posts copper, one iron (1). Silk packing can be seen round posts. (The John Humphreys Odontological Museum, University of Birmingham.)

teeth on to their pins. Many English dentists use no other means to secure artificial teeth to the plates (Fig. 5). He retains complete dentures by spiral springs and there is no mention of flat springs. His book contains a very valuable vocabulary describing the French technical terms [18].

Lefoulon makes some interesting suggestions, although his work of 1841 owes a great deal to Maury. When he uses human teeth, he prefers those of men slain in battle, in the full vigour of life from the eighteenth to the fortieth year (thus following Maury). Those chosen are pierced at the end of the root, arranged on a thread and kept in flax-seed. He describes ligatures in the same terms as Maury, and says that while they had been in use for a long time he considered them undesirable and he rarely used them. He states categorically that human teeth are daily employed with the greatest advantage. He suggests that to overcome the shrinkage which is inevitable when a porcelain denture is baked, a process used in statuary to enlarge or reduce a model might be employed to enlarge the model by say a tenth part [19].

A most detailed account of the making of artificial teeth in England is to be found in Robinson (1846). He describes gold dentures with English, French and American porcelain teeth, with the differences between them, and the carving of hippopotamus tusk to fit a model. A section of the tusk is cut into two halves and applied to the model so that the fibres are horizontal, unless the enamel is to be retained, when the fibres are placed vertically. This is in fact seen in dentures. The surface of the model is then hardened with wax and resin and painted with rose pink and oil. The block is repeatedly applied, and the high spots cut away until the denture fits. A second model is used when the first has lost its sharpness. Robinson mentions the machine invented by Tomes in 1845 but says it is not very satisfactory owing to the difficulty of producing undercuts. He then apparently painted a patient's gum with red paint and tried the plate in the mouth. He used springs only when the alveolus was shallow, since the firmness and steadiness of the plate depended entirely upon the accuracy of the fit. The methods and formulæ for baking porcelain teeth are given, Robinson saying they have always remained a profound secret in England, and he quotes from the American textbook of Goddard and Parker, 1844 [20]. French textbooks had given formulæ for many years. He gives instructions for baking several teeth in blocks, and this may be important in the dating of a porcelain denture, since a poorly fired example of *c.* 1850 may bear some resemblance to the work of de Chémant of *c.* 1800.

In the fifties a number of new materials were tried as bases for artificial teeth; I have mentioned Harrington's tortoiseshell process, but in addition there was gutta-percha first patented by Truman in 1848, buffalo horn, aluminium, the "cheoplastic" metal of Blandy, and of course vulcanite.

An important lecture by Robert Hepburn at the London School of Dental Surgery in 1864 shows the position of mechanical dentistry at that time. He says that metallic and vegetable substances were gradually superseding bone work. He evidently was convinced that ivory still had an important part to play, especially for lower dentures, for orthodontic plates and for temporary dentures. He says that ivory was still much used for side blocks, and that there were practitioners in London and Paris who still used no material as a base but bone. It was usual to send hippopotamus tusks to grinding mills, where the enamel was ground off at a cost of from two and sixpence to five shillings each. Mineral teeth may be cemented on their pins with sulphur. He gives a formula for staining gums pink, and states that the denture must be placed in the boiling solution for a few minutes, the parts to remain white being protected with plaster of Paris [21].

The identification of the particular ivory used may be difficult owing to the smallness of the specimen or the amount of destruction of the surface that has taken place. There is a very wide colour variation, ranging from almost black through every shade of yellow and brown to almost pure white. The colour, therefore, is of no value in identification, as it appears to depend on the smoothness of the denture originally, the care with which it was cleaned and the action of the saliva.

I have examined a total number of 473 dentures from various sources, either consisting entirely of ivory or containing a substantial proportion, and

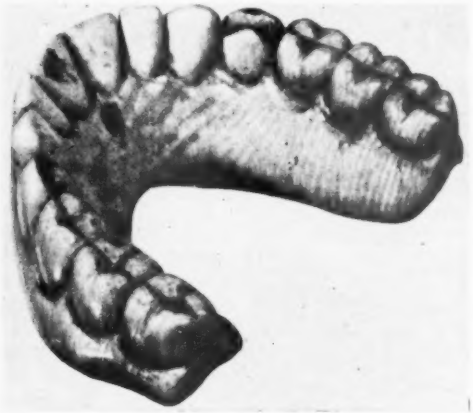

FIG. 6.—Upper denture, elephant ivory with typical striations.
(The Museum of the British Dental Association.)

of this number 269 were made of hippopotamus, 146 of walrus, 5 possibly of elephant, and 22 doubtful. I have also seen 4 wooden and 27 porcelain dentures.

The materials of choice when an ivory denture was to be made were elephant ivory, hippopotamus ivory, sperm whale, walrus ivory, and bone, and it is necessary to be able to distinguish them. If the denture is small, say one or two teeth, this may be a matter of considerable difficulty or even impossibility, since it is a necessity that the identification must not damage the denture in any way, otherwise a microscopic examination of a small fragment would be decisive. The denture examined must be cleaned and slightly polished and a lens of about six magnifications is suggested. The method employed is to obtain specimens of various ivories, cut and polished so that three planes can be examined. The reason for this is that each ivory possesses a definite structure which is visible on sometimes one only of the cut surfaces and it is thus possible to identify the material of the denture under examination by comparison.

Elephant ivory.—The form and size of the elephant tusk is too well known to require description. A longitudinal section shows an apparently structureless outer layer of cementum perhaps $\frac{1}{8}$ in. in depth, known to ivory workers as the rind. Next there is a layer of denture marked by longitudinal lines which become more indefinite as the centre of the tusk is approached. Finally the lines tend to disappear towards the centre and the ivory appears to be structureless. In transverse section the cementum appears to be structureless, but the outer layers of the dentine present a very typical criss-cross appearance which is difficult to describe but enables a denture made of elephant ivory to be identified with certainty (Fig. 6). The "lines" described no doubt indicate the periodicity of deposition of calcific material of varying density.

Hippopotamus ivory.—The canines were usually the teeth used, and normally the lower. The upper canines are comparatively small and only a small portion is erupted, while the lower are very large and weigh from $2\frac{1}{2}$ to 10 lb. The outer surface of the tusk is covered with enamel, the rest being covered by cementum. A longitudinal section in one plane shows no apparent structure, but the longitudinal plane at right angles to this shows a series of longitudinal lines which apparently diminish as the centre of the tusk is approached. A transverse section also shows these lines parallel to the surface and hence concentric. They are narrower and more closely packed than in the elephant, and there is absolutely no suggestion of the "criss-cross" appearance. Robinson states that if natural or mineral teeth are to be inserted in the piece, the tusk must be applied horizontally to the model. On the occlusal surface, therefore, the lines would not be apparent, but would be visible on the buccal surface (Fig. 7). Occasionally dentures of hippopotamus tusk are seen with the enamel on the labial surface of the artificial teeth. The enamel is usually "crazed," that is showing very numerous cracks, and is often of a curious blue colour (Fig. 8). Robinson (among other authors) notes this and considers that the enamel covering is best left on the sides of the denture for this reason. When the enamel is retained the tusk is

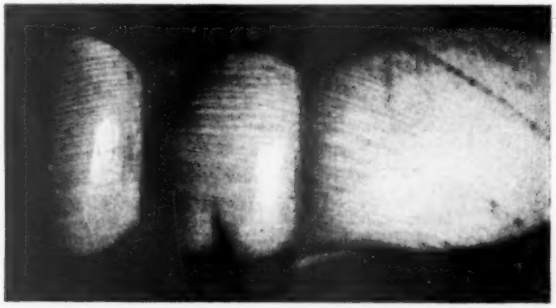

FIG. 7.—Partial upper denture, hippopotamus ivory, buccal view showing typical striations. × 3 approx. (The Fauchard Museum, Paris.)

FIG. 8.—Partial upper denture, hippopotamus ivory with enamel on labial surface. Retained by silk ligatures, *c.* 1800. (The John Humphreys Odontological Museum, University of Birmingham.)

FIG. 9.—Upper denture, walrus ivory showing secondary dentine. Made in Leamington, *c.* 1870. (Collection of the author.)

cut transversely and consequently the lines are visible on the occlusal surface.

Walrus ivory.—This is obtained from the enormously developed upper canine teeth. A transverse section of a walrus canine shows a layer of cementum which is structureless, a layer of dentine which is structureless and a mass of secondary dentine which has a very typical appearance and enables ivory of this animal to be identified. This secondary dentine occupies a great part of the body of the tooth, even when the tooth comes from a young animal, and appears to be more translucent than the true dentine, consisting of rounded masses set in the whiter dentine. A large number of dentures have been identified as having been made from this ivory, and in each case identification is possible because fragments of the secondary dentine are seen (Fig. 9). Nasmyth (1839) says "This substance . . . is the cause why only the outer shell of the canine teeth of the walrus is applicable for finer bone-work." [22.]

Sperm whale—The teeth of this animal are of poor colour, and a transverse section shows secondary dentine of a very dense type, quite different in appearance from that of a walrus. Hepburn says "As the teeth of the whale and the tusks and teeth of the wild boar have fallen into disuse, I need say nothing about these animals." He goes on to say that he has frequently used the small grinding teeth of the boar for side blocks and found they were very satisfactory. I have not seen any dentures constructed from a whale's tooth.

Bone.—This appears to be structureless when examined, and a tentative identification of this material can only be made when the preceding materials are eliminated.

In an effort to obtain a more scientific and precise method of identification than slight magnification of the surface, I endeavoured to obtain the specific gravity of elephant ivory, hippopotamus ivory, walrus ivory and bone from the hock of a cow, the results being elephant

ivory 1·78, hippopotamus 1·97, walrus 1·92, bone 2·08. Several specimens were tested in each case and these figures represent the average. Two small hippopotamus dentures were tested and the specific gravity proved to be 1·92 in one case and 2·03 in the other, so that it would appear that though the figures are somewhat inconclusive, the method might be of value in some cases, the first necessity being the determination of the specific gravity of a large number of specimens of different ivories, using perhaps more precise methods than I have been able to employ. If the denture to be identified is small and presents no recognizable features in the way of striations, it must be confessed that the material of which it is composed cannot be identified without microscopic examination.

When I began investigations on the specific gravities of ivories I was not aware that any previous worker had had the same idea but in 1849 Alexander Nasmyth published tables showing the specific gravity and chemical composition of various sorts of ivory and of enamel. The actual chemical work involved was done by Thomas Thomson, Regius Professor of Chemistry at Glasgow, and his nephew, R. D. Thomson. The specific gravity of elephant ivory was 1·728–1·794, walrus 1·909, hippopotamus 1·866 [23]. I am not aware of any more recent work along these lines, but since these men were expert and experienced chemists, it may be assumed that these figures are accurate. The figures approximate my own results and it would appear that while the specific gravities of different ivories can hardly be used as a basis for identification, it is a matter which requires further research before a certain conclusion can be reached.

From the middle of the nineteenth century the dental supply houses advertised hippopotamus, walrus and whale teeth. Messrs. Claudius Ash have kindly given me some information relating to the period 1851–1875. During that period they offered hippopotamus teeth at 3/6 to 14/- per lb., blocks with enamel ground off 3/- to 25/- each, walrus tusks 2/6 to 4/- per lb., blocks 1/- to 7/- each. I understand that Messrs. Claudius Ash supply hippopotamus teeth to some dental schools for practice in the cutting of cavities, and thus hippopotamus is the oldest material in present use, having been used certainly from the end of the seventeenth century.

From the earliest times retention of removable dentures was by gold or silver wire, or silk or flax thread. As I have shown, the dentures illustrated by Paré are so retained, and a similar method is shown in the works of Fauchard [12], Bourdet [13], de Chémant [15], Gariot [16], Delabarre [24], Maury [18] and many others. Gariot's work of 1805 [16] was translated into English in 1843, and a footnote by the editors states "the practice of fastening one or more artificial teeth with ligatures to the adjoining teeth has long since been done away with." Lefoulon, writing in 1841, considered that ligatures are undesirable. It would be reasonably safe to say that a denture needing ligatures for retention would date before about 1825, although Robinson says in 1846 that the ligatures frequently employed to fasten dentures soon loosen the adjoining teeth.

Bands and wires round teeth are illustrated by Campani [25], by Delabarre [24] and most of the later authors. The wire bands and cribs of De la Fons [26] are a great improvement. There are considerable difficulties in attaching satisfactory gold bands or wires to ivory dentures, and this has been surmounted in a most ingenious way in a denture in the Odontological Museum of the Royal College of Surgeons, the band being in two parts, each separately inserted into the denture.

Another method of retention was by means of wooden pegs driven into the side of the artificial tooth to wedge it in place. This appears to be rare and I am able to show a specimen by the courtesy of the Director of the Fauchard Museum, Paris (Fig. 10).

Campani [25] illustrates three dentures replacing a number of upper incisors which appear to be retained by a pair of clips which fit over the alveolus.

Complete dentures were always retained by springs and in the eighteenth century these were usually flat and were inserted in the posterior surface of the denture behind the last molar tooth. Campani [25] and Arroyo [27] illustrate this. Laforgue [28] illustrates this type, but also an upper denture attached to a lower frame on

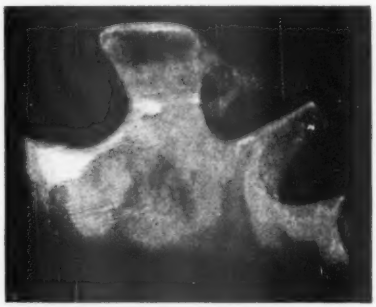

FIG. 10.—Upper denture, hippopotamus ivory, retained by two wooden pegs. (The Fauchard Museum, Paris.)

which the springs are fastened at the side of the molars (Fig. 11), and henceforth springs are shown in this position. It would appear, therefore, that springs fastened behind the last molar tooth would indicate an eighteenth century date. Fauchard says that a complete upper denture can be held by the sole support of the cheeks and lower teeth, but it is only intended for ornament and pronunciation. However, it can be used for eating as he has seen.

The use of springs declined from about the middle of the last century, when more accurate impressions were obtained, but they continued to be used occasionally until very recently.

The dating of carved dentures may present great difficulties. If porcelain incisors are used dating is much simplified, because the teeth used are either tube teeth with gold or platinum tubes and consequently *c.* 1837 or later, or facings which cannot be earlier than 1808 when Fonzi introduced separate porcelain teeth. It is when the dentures are carved from one piece or when human incisors are used that the greatest difficulty arises. Early writers from Albucasis onwards mention the use of animals' teeth and ox bone, presumably for the incisors only; dentures of this type are rare, but when found they may well be assumed to be dated before about 1750, and possibly much earlier.

The value of a denture whose date can be determined within 25 years or so is immense, and if any such dentures exist in a museum they should be accurately labelled. In this connexion the dentures excavated at St. Bride's and preserved in the Odontological Museum of the Royal College of Surgeons are valuable.

Rath [29] illustrates upper and lower dentures stated to be from a Swiss tomb of *c.* A.D. 1500 and made from the femur of an ox with a strip of tin to act as a spring between the dentures. The molar portions are joined to the incisal block with wire. Presumably the date is correct, but it is hardly possible that tin would be used as a spring since it is so soft a metal. And in this connexion I would plead for a really accurate description of a denture used as an illustration, stating the material, whether the pins are gold, platinum, silver or base metal, and whether the post is smooth or threaded. If all the incisor teeth are present this information can be obtained by an X-ray which would also show if the pins fitted accurately or whether silk thread was used as a packing. Finally, if a tentative date is assigned, the reasons should be given.

It is tempting to believe that the cruder the workmanship, the earlier the denture, but this view may lead to difficulties and errors. It is possible to date certain dentures with some accuracy, either because it is known who made them, or for whom they were made: these can serve as models from which other examples can be dated, but it is essential to record as many as possible of such accurately dated specimens.

When springs or bands are present they may give valuable clues as to the date of manufacture by comparing them with illustrations from text-

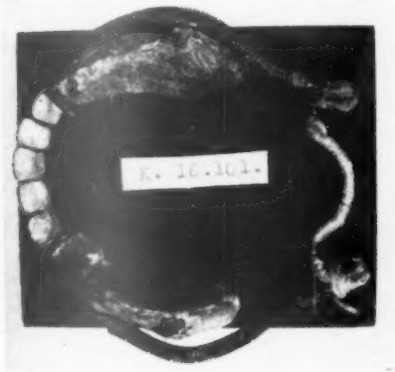

FIG. 11.—Upper denture, spiral springs attached to a curved, padded piece of metal which presumably fitted behind the lower incisors. The teeth are not human and the molar region is built up with resin or mastic. Late eighteenth century. (The Odontological Museum of the Royal College of Surgeons.)

FIG. 12.—Upper denture, grey porcelain, unglazed, *c.* 1820? (The Wellcome Historical Medical Museum, No. R.28370.)

books of various periods. Swivels may be hand-made or shaped in a press and it may be assumed that the former are the earlier.

Dating of porcelain dentures presents difficulties also. As I have said, de Chémant made all-porcelain dentures and the materials from which he made them are given in his patent of 1791. But the position is complicated by the fact that from about 1820–1850 many dental practitioners experimented with porcelain in France, in this country, in Ireland and in America, many textbooks giving formulæ (Fig. 12). As a matter of interest I have in my collection a copy of a letter written by John Parish of Bath dated 1812 in which he thanks de Chémant for his "Ratellier" [set of teeth] and says they "answer perfectly." He also sends a remittance of forty guineas.

If the denture is made of some unusual material, say tortoiseshell or gutta-percha, the dating is simplified, but it must be emphasized that almost all writers from Fauchard to Robinson stressed the usefulness of hippopotamus tusk over other materials, and therefore, in the absence of datable features, the date of construction of a hippopotamus ivory denture may well be a matter of conjecture.

[The lecture was illustrated by specimens, photographs, lantern slides and photostats of English patents relating to artificial teeth 1791–1863.]

Acknowledgments.—I am indebted to many friends for assistance in the preparation of this lecture but especially to the following: Dr. Lilian Lindsay; Dr. L. J. Cecconi, Curator of the Fauchard Museum, Paris; Mr. J. A. Donaldson, Honorary Curator of the Museum of the British Dental Association; Professor A. B. McGregor and Dr. E. A. Marsland, John Humphreys Odontological Museum, University of Birmingham; Professor A. E. W. Miles, Honorary Curator of the Odontological Museum of the Royal College of Surgeons of England; Dr. E. A. Underwood, Director of the Wellcome Historical Medical Museum; Mr. G. A. Johnson, Mr. C. Redmond and the photographic staff of the Wellcome Historical Medical Museum; M. Georges Dagen; Mr. A. F. A. Drake; Mr. A. F. E. Friedlein; Messrs. Claudius Ash, Sons and Co. Ltd.; and the Librarians of the Royal College of Surgeons and of the Medical School, Birmingham.

REFERENCES

1 JOHNSTONE, M. A. (1932) *Ann. Archæol. Anthrop.*, **19**, 121.
2 WAITE, W. H. (1885) *Indep. Practit.*, **6**, 195.
3 WEINBERGER, B. W. (1948). An Introduction to the History of Dentistry. St. Louis; **1**, 206, 370.
4 PARÉ, A. (1575) Les Œuvres. Paris; p. 718.
5 JOHNSON, T. (1649) The Workes of that famous Chirurgion Ambrose Parey. London; p. 578.
6 LINDSAY, L. (1933) A Short History of Dentistry. London; p. 45.
7 GUILLEMEAU, J. (1597). The French Chirurgerye newly translated out of Dutch into English by A. M.; Dort.
8 LINDSAY, L. (1926) *Brit. Dent. J.*, **47**, 1010.
9 COOKE, J. (1676) Mellificium Chirurgiæ: or, the Marrow of Chirurgery. London; p. 751.
10 LOWE, P. (1654). A Discourse of the Whole Art of Chyrurgery. 4th ed. London; p. 194.
11 NUCK, A. (1696). Operationes & Experimenta Chirurgica. *Lugd. Bat.*; p. 68.
12 FAUCHARD, P. (1728) Le Chirurgien Dentiste, Paris. Translated from the second edition of 1746 by Lilian Lindsay (1946). London, **2**, 75–98, 114–117.
13 BOURDET, E. (1757) Recherches et Observations . . . du Dentiste. Paris; **2**, 211–299.
14 BERDMORE, T. (1768) A Treatise on the Disorders and Deformities of the Teeth and Gums. London; pp. 261–267.
15 DE CHÉMANT, DUBOIS (1797) A Dissertation on Artificial Teeth in general. London; p. 20.
16 GARIOT, J. B. (1805) Traité des Maladies de la Bouche. Paris; pp. 295–323.
17 DAGEN, G. (1926) Documents pour Servir à l'Histoire de l'Art Dentaire. Paris; p. 218.
18 MAURY, F. (1828) Traité complet de l'Art du Dentiste. Paris; pp. 271–340.
19 LEFOULON, J. (1841) Nouveau Traité . . . de l'Art du Dentiste. Paris; pp. 329–431.
20 ROBINSON, J. (1846) The Surgical, Mechanical, and Medical Treatment of the Teeth. London; pp. 173–297.
21 HEPBURN, R. (1864) *Brit. J. dent. Sci.*, **7**, 453–467.
22 NASMYTH, A. (1839) Researches on the Developement, Structure, and Diseases of the Teeth. London; p. 91.
23 —— (1849) Researches on the Developement, Structure, and Diseases of the Teeth. London; pp. 206–214.
24 DELABARRE, C. F. (1820) Traité de la partie mechanique de l'Art du Chirurgien-Dentiste. Paris.
25 CAMPANI, A. (1786) Odontologia . . . Firenze.
26 DE LA FONS, J. P. (1826) A Description of a New Patent Instrument . . . Method of Fixing Artificial Teeth. London.
27 ARROYO, F. P. (1799) Tratado de las Operaciones . . . en la Dentadura. Madrid.
28 LAFORGUE, L. (1802) L'Art du Dentiste. Paris.
29 RATH, G. (1958) *Ciba Symp.*, **6**, 9.

Section of Psychiatry

President—ELIOT SLATER, M.D.

Meeting
March 10, 1959

A DEBATE was held on the Motion **That Most Mental Hospitals Meet the Needs Neither of Their Patients Nor of the Communities Which They Serve.**

The Motion was proposed by Dr. JOSHUA BIERER and Dr. ARTHUR POOL, and was opposed by Dr. A. A. BAKER and Mr. KENNETH ROBINSON, M.P.

Subsequent speakers included Dr. NOEL HARRIS, Dr. D. H. CLARK, Dr. SAWLE THOMAS, Dr. DONALD BLAIR, Dr. D. C. WATT, Dr. M. E. F. WOOLLASTON, Dr. S. L. LAST and Dr. R. FREUDENBERG.

The Motion was defeated by 24 votes to 22; there were a number of abstentions.

A short report of this Debate appeared in the *Lancet* (1959) i, 663.

Meeting
April 14, 1959

DISCUSSION ON THE ÆTIOLOGY OF MENTAL DEFECT [*Abridged*]

Dr. Brian H. Kirman (London):

Some Social Considerations

There are two main groups of factors in the genesis of mental defect: (1) Agents producing imperfect development of, or gross damage to, the brain, e.g. factors causing hydrocephalus, and events at delivery resulting in birth injury. (2) Unfavourable circumstances leading to a failure to realize potential mental capacity, i.e. an adverse material and cultural environment in childhood.

Both groups of factors are important in each individual. Consider the case of a boy with meningovascular syphilis, which left him hemiplegic and mentally retarded: he was sent to hospital not only because of his retardation, but also because he was illegitimate and homeless. Early residence in an institution interferes with verbal development and other accomplishments. At 11 years, his Wechsler I.Q. was 57 with a performance achievement of 75, the latter being equivalent to a mental age of 8 years. However, his reading age, on Burt's recognition test, was 4–6 years, and his arithmetic level was 6½ years on the Southend mechanical test. Though the boy's brain was damaged by the disease process, the unfavourable environment increased the backwardness.

In cases with severe defect, gross brain pathology is the rule and the prime reason for the mental defect. In a large post-mortem series (Crome, 1954) the overwhelming majority of brains showed obvious abnormality. In the educationally subnormal or feebleminded, on the other hand, upbringing and social factors are of greater ætiological importance. Thus, a child with severe kernicterus, resulting in crippling athetosis and marked deafness, will progress little even in an optimum environment. If, however, the child is only doubtfully defective, a suitable environment may tip the balance favourably.

Educational opportunity.—Even above imbecile level many patients show gross organic brain pathology. An example (Cowie, 1951) is a young man, who has done well at special school (Cowie and Brandon, 1958), with normal intelligence despite phenylketonuria. However, the intellectual level and social attainment of subnormal individuals is a function of the circumstances of their upbringing. It is difficult to separate individual factors from the general social and cultural environment, but it is possible to deal with particular aspects.

Simple lack of educational opportunity is a handicap in homeless or institutionalized children. Much depends on the institution, but such findings are common. Mrs. Beatrice Fliess-Hermelin (Kirman, 1958) showed that children discharged to special schools improved in educational attainments, while those remaining in hospital did not. Lyle (1959) found that children attending occupation centres in hospital are more retarded than those attending daily from home. It may be objected that there is a distinction between mental deficiency and

educational retardation. In practice, any such distinction is nebulous. Educational retardation is part of, and aggravates, mental defect.

Uneven mental development.—The unreliability of predictions based on mental assessment of young children in general is shown in Table I (Jones, 1954, modified).

TABLE I.—CORRELATION COEFFICIENTS FOR RETESTS AT THREE-YEAR INTERVALS

Age in years	Ebert and Simmons (1943)	Honzik *et al.* (1948)
2 and 5	—	0·32
3 and 6	0·56	0·57
4 and 7	0·55	0·59
5 and 8	0·70	0·70
7 and 10	0·76	0·78
9 and 12 or 13	—	0·78

Part of the variation is technical in that a "reliable" assessment is difficult in young children. Real differences in speed and pattern of mental growth do occur, as shown in longitudinal studies. Apart from different rates of physiological maturation, much of this variation reflects changes in the intimate environment of the child during early development. Such changes influence the final outcome where a child is near the border of mental deficiency.

Family size.—There tends to be an inverse relationship between family size and intelligence (Scottish Council for Research in Education, 1949). It may be contended that this depends on genetic factors but the alternative hypothesis of the effect of reduced opportunity in a large family is attractive. We cannot make a contribution on this topic as our patients are young, their sibships incomplete and controls difficult to arrange. Penrose (1938) showed that parental fertility in his series was maximal at an I.Q. range of 80–90. The number of siblings in the Colchester Survey, however, was not greater for the feebleminded (3·7) than for idiots and imbeciles (3·9) (Penrose, 1954). Penrose states that the figures in the Colchester Survey are in excess of those in the general population (deaths in infancy are taken into account). In many of our patients admission to hospital and ascertainment as defective has been precipitated by the needs of the siblings and housing problems.

Adequacy of home.—Burt (1955) established that educational backwardness as defined by him was much commoner in the poorer boroughs of London. Penrose showed that the homes of feebleminded patients were subnormal, compared with a normal distribution for the homes of low-grade patients. An adverse early environment was a factor in 44 children admitted to the Fountain Hospital Group as mentally defective and subsequently discharged as educable (Craib and Woodward, 1958). This also applies to the period of adolescence, at which time dullards with inadequate homes are most liable to be dealt with as mental defectives.

The Clarkes (Clarke, 1957; Clarke *et al.*, 1958) showed that the environment influences the way in which dullards are dealt with in society, and that intelligence itself in dull adolescents may change considerably. Those with an adverse background gained more on standard intelligence tests. These increases were not artifacts and occurred at an age when such growth is thought normally to have ceased.

There is an undue proportion of illegitimacy among institutionalized mental defectives (Hilliard and Kirman, 1957), 7·1% for a series of 350 defectives, compared with 5·4% for maternities in the general population in the South-Eastern area. The real discrepancy is probably greater because of the differential stillbirth and infant mortality rates. Illegitimacy has an adverse effect beginning before birth and continuing into adult life. It is likely to predispose to classification as mentally defective during the pre-school period, when the homeless borderline child may be classed as ineducable, and the school-leaving period, when the child from a residential school may be thought to need institutional care. Homeless dullards brought before the courts are more likely to be dealt with as mental defectives.

Craib and Woodward (1958) found that 11 of 42 children suitable for trial at school were illegitimate, a much higher rate than for the whole hospital. Of 9 children in the series who were subsequently again excluded from school, 5 were illegitimate.

Hereditary factors.—Brandon (1957) found the average I.Q. of 108 children born to mentally defective women to be of the order of 91. Only 4 children scored below I.Q. 65, despite 80% of the children being illegitimate. The mothers in Brandon's study were not gross defectives, their I.Q.s averaging 73·5. But they are a fairly typical sample of mentally defective parents, showing that most children of defectives are normal. Those children who were brought up by their own parents did better than those who were not.

The considerations mentioned emphasize the fluidity of mental development and its susceptibility to environmental influence. Important adverse factors which may tip the balance in favour of mental deficiency in the borderline group are early segregation and an inadequate home with poor social circumstances.

REFERENCES

BRANDON, M. W. G. (1957) *J. ment. Sci.*, **103**, 710.
BURT, C. (1955) The Subnormal Mind. London; p. 123.
CLARKE, A. D. B. (1957) *J. Midland ment. Defic. Soc.*, No. 4, p. 58.
——, CLARKE, A. M., and REIMAN, S. (1958) *Brit. J. Psychol.*, **49**, 144.
COWIE, V. A. (1951) *Lancet*, i, 272.
——, and BRANDON, M. W. G. (1958) *J. ment. Defic. Res.*, **2**, 55.
CRAIB, M. F., and WOODWARD, M. (1958) *J. ment. Sci.*, **104**, 115.
CROME, L. (1954) *J. ment. sci.*, **100**, 894.
EBERT, E., and SIMMONS, K. (1943) *Monogr. Soc. Res. Child. Developm.*, **8**, 113.
HILLIARD, L. T., and KIRMAN, B. H. (1957) Mental Deficiency. London; p. 71.
HONZIK, M. P., MACFARLANE, J. W., and ALLEN, L. (1948) *J. exp. Educ.*, **18**, 309.
JONES, H. E. (1954) In: Manual of Child Psychology. Ed. L. Carmichael. 2nd edit. New York; p. 637.
KIRMAN, B. H. (1958) *J. ment. Sci.*, **104**, 167.
LYLE, J. (1959) In preparation.
PENROSE, L. S. (1938) A Clinical and Genetic Study of 1,280 Cases of Mental Defect. London; p. 62.
—— (1954) The Biology of Mental Defect. London; p. 54.
Scottish Council for Research in Education (1949) The Trend of Scottish Intelligence. London; p. 101.

Dr. J. M. Berg (London):

Some Pathological Factors[1]

The following account of some of the pathological conditions operative in mental defect is based on a survey of 1,900 patients admitted to the Fountain Hospital Group in the past twelve years. Seven-eighths of these are idiot or imbecile (low-grade) children, the remaining eighth being mainly adults in the feebleminded (high-grade) range. Females predominate slightly.

[1]This material constitutes part of a paper awarded a prize in the South-West Metropolitan Regional Hospital Board's 1958 competition for research reports.

In a group like this, composed largely of low-grade patients, physical signs indicative of brain lesions are common. For example, 668 of the 1,900 patients (35%) had one or more of the following 4 findings (% incidence in brackets): epilepsy observed after admission to the Fountain Hospital (21%), cerebral palsy (19%), microcephaly (8%), and hydrocephalus (4%). Despite the frequency of such physical signs, and the fact that the vast majority of brains of low-grade defectives show gross abnormalities at post-mortem, ætiological factors and distinct syndromes are only established in a small proportion of cases. Table I shows those found among the present 1,900 patients.

A brief commentary on the data presented in Table I follows:

Mongolism, by far the commonest single syndrome noted in patients admitted to the Fountain Hospital Group, has a relatively high incidence (13·1%) in this series because of the predominance of young low-grade patients. It is, however, not uncommon in the general population. An estimated figure, for all ages, is almost 1 in 3,000 (Penrose, 1949). The incidence may now be higher, for respiratory and other infections to which cases of mongolism are prone, and which carried a considerable mortality, generally respond well to antibiotics.

Despite many established facts, the cause of mongolism remains an enigma. A recent finding of great potential importance is that mongolism appears to be associated with the presence of an extra chromosome (Jacobs *et al.*, 1959).

TABLE I.—ÆTIOLOGICAL FACTORS AND DISTINCT SYNDROMES IN 1,900 MENTAL DEFECTIVES

Finding	No. of cases (% incidence in brackets)			Sex distribution	
	Likely or certain	Possible	Total	M.	F.
Mongolism	249 (13·1)	0	249 (13·1)	135	114
Meningitis	34 (1·8)	6 (0·3)	40 (2·1)	20	20
Phenylketonuria	28 (1·5)	0	28 (1·5)	14	14
Kernicterus	21 (1·1)	0	21 (1·1)	14	7
Tuberous sclerosis (epiloia)	15 (0·8)	0	15 (0·8)	6	9
Encephalitis	6 (0·3)	9 (0·5)	15 (0·8)	7	8
Maternal rubella	8 (0·4)	2 (0·1)	10 (0·5)	6	4
Congenital syphilis	8 (0·4)	0	8 (0·4)	3	5
Cerebral lipoidosis	7 (0·4)	1 (< 0·1)	8 (0·4)	6	2
Cretinism	2 (0·1)	3 (0·2)	5 (0·3)	2	3
Postnatal head injury	2 (0·1)	3 (0·2)	5 (0·3)	4	1
Sturge-Weber syndrome (nævoid amentia)	4 (0·2)	0	4 (0·2)	1	3
Von Recklinghausen's disease (neurofibromatosis)	4 (0·2)	0	4 (0·2)	1	3
Infantile gastro-enteritis	1 (< 0·1)	3 (0·2)	4 (0·2)	2	2
X-ray irradiation during pregnancy (child defective)	1	0	1	0	1
Pertussis immunization	1 (0·2)	0	1	1	0
Galactosæmia	1	0	1	1	0
Hypoglycæmia	1	0	1 (0·4)	1	0
Thiouracil treatment during pregnancy (child defective)	0	1	1	1	0
Neonatal septicæmia	0	1 (0·2)	1	1	0
Sagittal sinus thrombosis	0	1	1	0	1
Lead poisoning	0	1	1	1	0
Totals	393 (20·7)	31 (1·6)	424 (22·3)	227	197

Meningitis.—In the Colchester survey (Penrose, 1938), no more than 8 of the 1,280 cases (0·6%) appeared to owe their defect to meningitis. The incidence in this series is at least 1·8%. This difference may partly be due to differences in the sample of patients under review, but part is probably due to a greater survival rate from meningitis following the introduction of antibiotics. This has resulted in an overall increase of survivors with persisting mental and physical morbidity. It is notable in this connexion that, of the 34 cases where meningitis was a likely or certain cause of the mental defect, 17 were instances of tuberculous meningitis, a disease which was practically always fatal before the advent of chemotherapy. Six others were due to the meningococcus, 5 to the pneumococcus, 2 to *H. influenzæ* and 1 to *B. pyocyaneus*. The remaining 3 were acute purulent meningitides in which the causal organisms were not isolated.

Phenylketonuria. — Compared to only 2 instances of the disease recorded in the Colchester survey, phenylketonuria is relatively common in this series. Jervis (1952), from America, quoted 0·5 to 1·0% for the incidence of phenylketonuria among idiots.

One of our cases had an I.Q. ranging from 76 to 97 on different tests (Cowie, 1951). This is unusual, most phenylketonurics being low-grade defectives. Detection of the heterozygous carriers of the disease (Hsia *et al.*, 1956) and low phenylalanine diets (Woolf *et al.*, 1958) respectively hold out hopes of prevention and effective treatment.

Kernicterus.—Of the present 21 cases, 18 were due to rhesus factor incompatibility, 1 was attributed to prematurity and 2 were of obscure origin. Besides such serious consequences of hæmolytic disease as noted here, slight impairment in average intelligence has been reported in children who had apparently recovered from erythroblastosis fœtalis without evident motor disability (Gerver and Day, 1950).

Tuberous sclerosis, Sturge-Weber syndrome and neurofibromatosis may be considered together for, though each is generally distinct, some overlap between them sometimes occurs. The extent and degree of cerebral involvement varies considerably, so that all ranges of mental impairment are noted. In some cases mentality is normal.

Encephalitis.—Two of the present cases were associated with measles and 1 each with chickenpox, whooping cough and vaccination. A sixth was thought to have encephalitis lethargica. In none of these was the diagnosis certain. 6 of the remaining cases were considered by Bourne (1955) to be examples of epileptic dementia rather than encephalitis. These comments perhaps reflect the difficulty of establishing encephalitis as a cause of mental defect.

Maternal rubella early in pregnancy is now a well-established cause of mental defect in the offspring of mothers thus infected. 8 of the cases in the present series have been analysed and discussed elsewhere (Kirman, 1955).

Congenital syphilis.—With the decline in the incidence of syphilis in this country (*Rep. Minist. Health, Lond.*, 1957), mental defect from this cause is becoming increasingly rare. In the Colchester survey, congenital syphilis was ten times more frequent than in this series—4·0% as opposed to 0·4% here.

Cerebral lipoidosis.—Of the 8 cases, 5 were instances of Hurler's disease (gargoylism), 1 had cerebromacular degeneration and 2 were unclassified. The diagnosis was uncertain in one of the unclassified cases. These conditions are usually regarded as recessively transmitted.

Cretinism.—Compared to the low incidence in this series, cretinism was diagnosed in 16 cases (1·25%) in the Colchester survey. The decline in the proportion of cases of amentia due to cretinism has been attributed to the beneficial results of treatment (Tredgold and Soddy, 1956). However, some reduction in thyroid activity is not uncommon in mental defectives (Kirman, 1957).

Postnatal head injury is probably a rare cause of severe mental defect because brain injury sufficient to produce such serious consequences is frequently fatal. Less gross forms of post-traumatic intellectual impairment are commoner and more difficult to assess.

Infantile gastro - enteritis. — Crome (1952), describing the findings in one of the present cases, suggested that prolonged dehydration is likely to have led to hæmoconcentration and stagnation of the circulation, and that the resulting anoxia may have been the leading cause in the production of the severe mental and physical handicaps. 3 other cases in this series may have had a like pathogenesis.

Other diagnoses in single cases (*see* Table I) were of probable ætiological significance in four cases and of possible significance in another four.

The above causes and syndromes account for

only some 22% of the 1,900 patients reviewed, a proportion reduced even further if one excludes syndromes which, though clear-cut, have an unknown cause (e.g. mongolism). Other such conditions are known, examples of which have not been found in this series, e.g. degenerative disorders like Schilder's disease, infections like maternal toxoplasmosis and metabolic disorders like Wilson's disease: such are, however, very rare. Much therefore remains to be elucidated before causation in the field of mental defect is anything like fully understood. Besides the vastly important role of psychological, social and cultural factors, which Dr. Kirman has dealt with, one may mention several other facets which seem to merit greater attention. These include: (*a*) Genetic studies of families of defectives. (*b*) The maternal state during pregnancy, particularly perhaps during its earlier organ-forming stages, in relation to frank disease, apparent health, diet, &c. (*c*) The birth process itself and the early neonatal period. Related to these questions are factors like the mode of presentation and delivery and the wide ramifications of the problems of multiple births and prematurity. (*d*) The physical aspects of the environment in which the growing child develops.

REFERENCES

BOURNE, H. (1955) *J. nerv. ment. Dis.*, **122**, 288.

COWIE, V. A. (1951) *Lancet*, i, 272.

CROME, L. (1952) *Arch. Dis. Childh.*, **27**, 468.

GERVER, J. M., and DAY, R. (1950) *J. Pediat.*, **36**, 342.

HSIA, D. Y. Y., DRISCOLL, K. W., TROLL, W., and KNOX, W. E. (1956) *Nature, Lond.*, **178**, 1239.

JACOBS, P. A., BAIKIE, A. G., COURT BROWN, W. M., and STRONG, J. A. (1959) *Lancet*, i, 710.

JERVIS, G. A. (1952) *Amer. J. ment. Defic.*, **57**, 175.

KIRMAN, B. H. (1955) *Lancet*, ii, 1113.

—— (1957) In: Mental Deficiency. Eds. L. T. Hilliard and B. H. Kirman. London; Chapter 14.

PENROSE, L. W. (1938) A Clinical and Genetic Study of 1,280 Cases of Mental Defect. London.

—— (1949) *J. ment. Sci.*, **95**, 685.

Rep. Minist. Hlth., Lond. (1957) Part II.

TREDGOLD, R. F., and SODDY, K. (1956) A Textbook of Mental Deficiency. 9th edit. London; Chapter 14.

WOOLF, L. I., GRIFFITHS, R., MONCRIEFF, A., COATES, S., and DILLISTONE, F. (1958) *Arch. Dis Childh.*, **33**, 31.

Observations on the Natural History and Genetics of Old Age Psychoses: A Stockholm Material, 1931–1937[1] [*Abridged*]

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SWEDEN is a particularly favourable country for long-term follow-up studies and family investigations, owing to the official registration of addresses, and of admissions to mental hospitals.

Follow-up is important in the study of old age mental disorders because, among other things, it throws light on the problems of classification, and the part played by organic cerebral disease. Affective and paranoid psychoses of late onset have a far better prognosis, over a two-year period at least, than senile or arteriosclerotic dementia, and are probably distinct entities (Roth, 1955). But later on in the course of "functional" psychoses signs of cerebral degeneration might still appear, and lead to premature death. I have thought it worth while, therefore, to present in brief a few results of a follow-up that is almost complete in two senses, since not only were all patients traced, but at the end of the investigation only 17 out of 236 were still alive. After a minimal interval of nineteen years the subsequent fate of these few survivors can hardly be relevant to the ætiology of the original psychosis.

Secondly, a *family investigation* was made. Genetical studies in involutional and old age mental disorders have produced conflicting results. Schizophrenia has been found among the relatives by some authors (Kallman, 1953), but not by others (Stenstedt, 1959); and whether the depressive forms are mainly endogenous or exogenous is uncertain. Clinical data are not always given. It seemed possible, therefore, that a genetical study closely related to clinical findings might succeed in clarifying some of the problems. Here I shall allude to personality, and to the frequency of certain exogenous factors, including physical illness.

The *material* was gathered at the Psychiatric

[1]Work done during the tenure of a Leverhulme Fellowship, Mental Health Research Fund, and carried out at the Psychiatric Clinic, Karolinska Hospital, Stockholm (Head, Professor T. Sjögren).

Hospital, Stockholm. The case-records of patients aged 60 years and over, admitted during the period 1931–1937, were examined, and all those with affective disorder, with paranoid psychosis of late onset (after the age of 59) (57 cases) or with senile or arteriosclerotic psychosis (82 cases) (the "dementia" group), were selected for follow-up. 6 cases probably belonging to one of these conditions could not be diagnosed and were discarded. No "mixed group" was formed. The remaining 106 cases had other diagnoses. The affective group was later subdivided into "early onset" (52 cases) and "late onset" (45 cases) groups (onset before and after 60 years old). The *diagnostic criteria* were based on those given by Roth (1955) but were probably rather wider. Cases were included that did not show the complete picture either of a depressive symptom-complex or of paraphrenia; but all those with dementia were assigned to the dementia group whether or not some functional symptoms were also present.

Follow-up.—Information was obtained from hospital records and from relatives, and the place and cause of death ascertained from official sources. The actual time survived in each group is compared with the expected survival time, based on the mean expectation of life for the Stockholm population of corresponding age; and the rate of mortality at various intervals after admission, as well as the relative frequency of different causes of death, are compared with expected values derived from the official statistics, after standardization for age and sex.

In the *dementia group* the time survived was only about one-fifth of that expected, and death was due in a high proportion of cases to "non-specific" causes (senility, myocardial degeneration, arteriosclerosis, bronchopneumonia) characteristic of old age. Cerebral hæmorrhage and other forms of cerebrovascular disease were given as the primary cause of death in only 15% of cases, and other specific conditions were also rare. Dementia in the aged is evidently associated with early death; and since this is true even in those cases where the physical state is well preserved, cerebral disease itself is mainly responsible. In the arteriosclerotic group vascular degeneration in other organs also plays a part.

Of all groups, the evidence for cerebral disease is least convincing among the *paranoid states*. The period of survival is almost normal, and death is due to causes similar to those in the general population. Late in the illness, and at an advanced age, signs suggestive of dementia may appear, but are difficult to distinguish from the deterioration seen in chronic schizophrenia. Many of the cases eventually presented this picture. It is also possible that schizophrenia potentiates the normal mental decline of old age.

The *affectives with early onset* survived for nine-tenths of the expected time. The increased mortality can be attributed in part to the effects of the psychosis, i.e. suicide, or exhaustion and inanition; but the appearance of focal signs of central nervous system disease in 12% of cases, together with 6 deaths (out of 46) attributed to cerebral hæmorrhage, provides some support for the suggested association between manic-depressive psychosis and arteriosclerosis.

The follow-up results among the *affectives with onset late in life* do not admit of any simple interpretation. The group as a whole survived for only seven-tenths of the normal span, but there was wide divergence in the prognosis within the group: one-quarter of the cases survived fifteen years or more, but of the 43 deaths two-fifths occurred within five years of admission (among those aged 70 and over the psychosis often appeared during the last few months of life), and among the survivors mortality continued to be above the normal. But cerebral arteriosclerosis does not seem to be an important factor in accounting for this increased mortality. An organic picture with dementia, in one case with focal signs, developed in only 2 cases, and only 1 other case showed signs of focal central nervous system disease, immediately preceding death from cerebral hæmorrhage. In 5 other instances death was attributed to cerebral hæmorrhage, but confirmatory clinical evidence was lacking. Naturally, cerebral arteriosclerosis cannot be excluded as a factor in some cases, perhaps 10%. The increased mortality was, however, probably due in the main to the presence of somatic disease of various kinds, which was relatively common in this group.

Personality: the paranoid group.—Follow-up has shown that somatic disease, including cerebral disease, is not likely to be an important ætiological factor in this group, the most striking characteristic of which is the predominance of unmarried women. Is it possible then, to attribute the illness wholly or in part to emotional deprivation and loneliness? Ødegaard (1953) holds that the predominance of single people among the mentally ill is largely due to selection, and not to protection afforded by marriage. This explanation would be difficult to accept among those falling ill for the first time late in life, unless some previous mental abnormality or personality deviation, reducing the chances of marriage, could be demonstrated.

Previous mental abnormality among the 57

cases was not frequent (3 cases with possible psychotic breakdown with quick recovery early in life and 4 others with emotional disturbances at the climacterium). Personality deviations were more impressive. 15 out of 33 female cases about whom information was available, i.e. 45%, were described in terms such as suspicious, peculiar, cold, hard, arrogant, jealous or shy and solitary. The incidence of these schizoid traits was significantly less among the affective disorders (18%). Adherence to minority religious groups was also much commoner among the paranoid cases. Of the 9 males, 6 were known to have been solitary, homosexual, criminal or vagrant.

Personality difficulties also presumably had a bearing on the fact that, of the females, over one-quarter had had illegitimate children, and that only 2 of them later married the fathers. Among the much smaller number with illegitimate children in the affective groups, the majority subsequently married. Most of these children were brought up separately from their parents and, at the time of admission into hospital, personal contacts were often greatly reduced, so that over one-third of admissions in the paranoid group were arranged by the police or other social agencies. These findings support the hypothesis of selection for marriage, and suggest that both the failure to marry and the "social isolation" eventually observed in many cases had a common origin in personality. In some, but by no means all cases, the psychosis itself can be seen as an exaggeration of pre-morbid personality traits.

Precipitating factors, such as acute physical illness, loss of members of the family or of close friends, and other factors causing psychological stress, such as quarrels within the family or economic difficulties, were all commoner among the *late onset affectives* than among the other two functional groups. Three-quarters of these patients had experienced stresses of some kind, compared with one-half among the early onset affectives, and only one-quarter in the paranoid group. Moreover, serious physical disease, of progressive or disabling kind, was found to be commonest in the group under consideration, and included ulcerative colitis, paralysis agitans, loss of sight, intermittent claudication and carcinoma of the stomach.

In many of these cases with affective disorder of late onset the ætiology seems to be multiple. Specific genetic factors play a role, as does personality, which often colours to a marked extent the clinical features of the illness. But extrinsic factors often appear to be more important than in the other groups. Cases where extrinsic factors were present often showed, not a sustained mood disorder of endogenous type, but features suggesting reaction to adverse circumstances. Such cases are often diagnosed as depression associated with cerebral arteriosclerosis, on the grounds of the fluctuating mood, particularly if there are signs of coronary or peripheral arteriosclerosis, as there often are. Here it is suggested that they should be regarded as reactive or symptomatic states, the contributory factors including personality, situational factors and alcoholism, as well as physical illness. The nature of the illness determines the prognosis for life; the higher incidence of serious somatic disease would account adequately for the increased mortality found in this group. Within the initial five-year period, conditions other than cerebrovascular disease caused almost three-quarters of the deaths, and there was no increase in the frequency of "non-specific" causes, such as is found in the dementia group. In its relationship to affective disorders of late onset cerebrovascular disease may be regarded in the same light as any other somatic disease.

Family investigation was confined to the functional groups. Table I shows the morbidity risks for schizophrenia and for affective disorder among all first-degree relatives (parents, sibs and children). Including all possible cases, there were 23 secondary cases of schizophrenia, of which 15 had been treated in mental hospitals. The late onset affective group and the paranoid group each contained 7 hospitalized schizophrenics among the relatives, both sexes being represented. Of relatives with affective disorder about half were hospital cases.

TABLE I.—MORBIDITY RISKS AMONG FIRST-DEGREE RELATIVES MINIMAL AND MAXIMAL FIGURES

	Affective disorder*	Schizophrenia†
Early affective group	10·0–12·7 ± 2·1	0·6 ± 0·4
Late affective group	3·5– 5·7 ± 1·4	2·5–4·2 ± 1·3
Paranoid group ...	2·9– 3·3 ± 1·1	3·6–5·6 ± 1·7
General population ..	1·0– 3·0	1·6

*Weinberg (1925), risk-period 20–60. †Weinberg (1925), risk-period 20–50. (Morbidity risks for schizophrenia exclude parents.)

The morbidity risks among the relatives in the *early onset group of affectives* clearly resemble those found by several authors for manic-depressive psychosis; there is an increase in secondary cases of affective illness, but not of schizophrenia. The same clear pattern is not seen among the relatives of the *late onset affective group*. It appears from this and from Stenstedt's (1959) work that the risk for affective disorder among relatives of probands falling ill for the first time at the age of 60 or over is 4–5%, a figure which is probably higher than expecta-

tion, but lower than the risk among relatives of manic-depressive probands. There is no genetic evidence for a specific type of affective psychosis of late onset since, among the relatives, cases with onset before the age of 50 predominated in both the two affective groups.

That manic-depressive psychosis may begin late in life is not in question. Kraepelin (1921) and Stenstedt (1952) both found that 6–7% of manic depressive patients fell ill for the first time after the age of 60. But the high incidence of physical illness and other stress factors found among cases of affective disorder arising in the senium, together with the relatively low morbidity risk among relatives, suggests that exogenous or symptomatic conditions are common. It is concluded that the group is heterogeneous, with endogenous depression accounting for probably less than half the cases. This accords also with the variability in the clinical picture and in the prognosis.

The *risk for schizophrenia* among the relatives in this group is not definitely increased (2·5–4·2%). Kallman (1953) gave a risk figure of 6% for schizophrenia among relatives of probands with involutional psychosis, but Stenstedt (1959) found no increase whatever among the relatives in a study of involutional melancholia. In the present material the finding of 11 schizophrenics (7 plus 4 doubtful) among the close relatives accords with the conclusion that the group is heterogeneous. Of the 10 probands with schizophrenic relatives, the majority had atypical psychoses, which in 3 cases had given rise to doubt about the most appropriate classification. 2 of these showed marked abnormalities of personality. The inclusion or rejection of these atypical cases among probands is likely to affect the results of genetical investigations of involutional and senile psychoses, and to account for some of the discrepancies found in the literature.

The *paranoid group* was relatively homogeneous, characterized by persecutory delusions and by hallucinations. The rather low morbidity risks for mental illness among relatives (3·6–5·6%) indicate that specific genetic factors contribute less to the occurrence of paranoid states of late onset than they do in the case of functional psychoses beginning earlier in life. Predisposition is certainly important, as is shown by the frequency of personality variants of schizoid type, and probably also by the preponderance of unmarried individuals. The adoption of secluded or eccentric modes of life is connected with the type of personality often found. But exogenous factors appear occasionally to be decisive for the development of a psychotic state, and cases occur where a personal relationship seems to have exerted a protective influence. Social isolation or long-standing emotional deprivation may bring the predisposition to light. The remarkable association with sex and civil state may be due in part to a social and cultural milieu that is particularly inimical to elderly spinsters.

Acknowledgments.—It gives me great pleasure to acknowledge the help and encouragement I received throughout from Professor Torsten Sjögren, Head of the Psychiatric Clinic of the Karolinska Hospital, Stockholm, and to record my deep indebtedness to Dr. Carl-Henry Alström for giving me all the many facilities of his Human Genetics Laboratory.

REFERENCES

KALLMAN, F. (1953) Heredity in Health and Mental Disorder. London.

KRAEPELIN, E. (1921) Manic-depressive Insanity and Paranoia. Edinburgh.

ØDEGAARD, Ø. (1953) *J. ment. Sci.*, **99**, 778.

ROTH, M. (1955) *J. ment. Sci.*, **101**, 281.

STENSTEDT, Å. (1952) *Acta psychiat., Kbh.*, Suppl. 79.

—— (1959) *Acta psychiat., Kbh.*, Suppl. 127.

WEINBERG, W. (1925) In : Handbuch d. sozialen Hygiene und Gesundheitsfürsorge. Edited by A. Gottstein *et al.* Berlin; Vol. **1.** *See also* LARSSON, T., and SJÖGREN, T. (1954) *Acta psychiat., Kbh.*, Suppl. 89, pp. 40-54.

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BOOK REVIEWS

A Manual of Anæsthetic Techniques. By William J. Pryor, M.B., Ch.B.(N.Z.), F.F.A. R.C.S. (Eng.), D.A.(Eng.), F.F.A. R.A.C.S. 2nd ed. (Pp. viii+228; illustrated. 27s. 6d.) Bristol: John Wright & Sons, Ltd. 1959.

This second edition within three years of the original publication is an indication of how well it has fulfilled its purpose of providing a practical handbook for the trainee anæsthetist.

As it is the less experienced practitioner who is most likely to be associated with accidents, it is very important that in a book designed for his guidance, due stress should be laid on all potentially dangerous situations. Dr. Pryor has not failed in his duty in this respect, but greater stress could have been laid on the hazard of vomiting in obstetric cases. While not every experienced anæsthetist can be expected to endorse all the details of the techniques described, the author has been careful to point out where the fields of controversy lie, and his main recommendations are unexceptionable. Such reservations are, of course, inevitable with any primer, and if appreciated, do not detract from its value. Many minor improvements have been made in the present edition of this well-produced volume, and it can be recommended to the anæsthetic house officers and junior registrars for whom it is intended.

Experimentation in Man. By Henry K. Beecher, M.D. (Pp. 80. 25s.) Oxford: Blackwell Scientific Publications. 1959.

The somewhat brutal and arresting title of this work is employed by its distinguished author to focus attention on the ethical problems involved in clinical trials.

Dr. Beecher is well qualified to write on this subject, and this thoughtful book—which is one of the American lecture series—is particularly concerned with those situations where the experiment or trial is not entirely, or even primarily, for the benefit of the individual patient concerned, but rather for the possible future benefit of patients in general.

The history of human experimentation is briefly outlined and the circumstances where it may justifiably be employed are defined.

The qualifications of the investigator and his relations with the subject are considered at length, as are the difficulties of selecting volunteers, whether they be patients or in normal health. Problems of consent and of understanding on the part of experimental subjects are also fully discussed.

The anomalies of the legal position are pointed out: for although many investigations enjoy moral support in the public or rather the professional eye, and every therapeutic procedure is, in a sense, an experiment, yet the legal doctrine remains that "a physician experiments at his peril".

In conclusion, the author reviews the many codes which have been drawn up for the guidance of the physician investigator. They range from Hippocrates to the Nuremberg Tribunals, and from Claude Bernard to the World Health Organization.

This discerning little treatise should be read, not only by those keen young registrars who feel that their professional advancement depends on their doing some clinical research, but also by many of their seniors who encourage them in that belief.

Christ and Freud. A Study of Religious Experience and Observance. By Arthur Guirdham, M.A., D.M., B.Sc.(Oxon.), D.P.M. (Pp. 193. 21s.) London: George Allen & Unwin, Ltd. 1959.

Freud regarded religion as a myth and Heaven as wish fulfilment. It is a part of the purpose of the author of this book to show that Freud's theories, although having some relevance to religions originating in Judaism, had little when matched against the religions of the Far East, such as Buddhism and Hinduism.

He maintains that neuroses are far less common in the practitioners of these dominantly emotional and self-annihilating religions than of the mainly intellectual ones of Christianity or Judaism, whose only true goal is some form of personal immortality after an indefinite period. This is largely due to the sense of sin which the theologians of these latter creeds inculcate into their followers, the amount of neurosis they engender depending on the emphasis placed on the sinfulness of man, the most prolific results being obtained by the narrower forms of Protestantism. Orthodox Christian theology and clericalism thus comes in for a lot of criticism as a breeder of neurosis.

The latter part of the book is largely devoted to the author's belief that there are many ways

by which true religious experience may be achieved, and to the thesis that "until we have learnt to see ourselves as nothing we will never liberate the real self which is buried, not only our social but our instinctive personality".

This is an interesting book with much in it to engender thought and arouse disputation but, although it is written with a pleasing style, it is not easy to read. The author expects too much knowledge of his readers and concedes too little in the way of intelligence. He thus assumes they have a knowledge of Freudian ideas, of the Testaments and of various religions other than Christianity which all do not possess, while at the same time he is frequently and unflatteringly repetitive.

General Pathology and Bacteriology for Dental Students. By Ronald L. Bishton, M.D. (Pp. viii+317; illustrated. 42s.) Bristol: John Wright & Sons Ltd. 1958.

The introductory chapter of this book consists of explanations of terms, and of some of the fundamental considerations of pathology and bacteriology. Chapters 2–5 deal with basic tissue responses, disturbances of circulation, and tissue death. There follow four chapters on general bacteriological subjects, including a section on sterilization, a chapter on antibiotics and chemotherapy and one on inflammation. The various groups of bacteria of medical importance, and the diseases they cause, are then dealt with systematically in twelve chapters, including chapters devoted to endocarditis and diseases of the lungs. The last three chapters deal with viruses, hæmatology and tumours. There is a brief bibliography.

The only criticisms to be made are on comparatively minor points. For example, the slide coagulase test, and the use of filter paper discs for antibiotic sensitivity tests, both probably the most widely used methods, are not mentioned. In the section on sterilization no mention is made of modern types of autoclave. In discussing antiseptics perhaps more should have been said about the advantages and disadvantages of the quaternary ammonium compounds since they are so commonly used at the present time.

Although not always going into great detail, this book is clearly and concisely written and covers the subject comprehensively. It is the author's intention to discuss bacteriology and general pathology in relation to each other, instead of treating bacteriology as an apparently isolated subject; he undoubtedly succeeds in this, and in relating both to the clinical aspect.

The book is to be recommended for undergraduate students, and could also be very useful to postgraduate students.

Cancer of the Skin. By John C. Belisario, C.B.E., E.D., M.D., Ch.M., D.D.M. (Pp. xvii+321+14; illustrated, 8 colour plates. 50s.) London: Butterworth & Co. (Publishers) Ltd. 1959.

Australian dermatologists have a wider experience of skin cancer than anyone else in the world and this excellent book by one of their leaders is most welcome. It is a blend of a full review of the literature with an account of the author's personal experience. The subject matter treated goes somewhat beyond the limits suggested by the title. Each form of cancer is described and discussed very clearly in all its aspects. By far the most valuable and authoritative sections are those on treatment. The accounts of the other conditions included in the book are sometimes less good. Thus, the section on histiocytoma does not mention that some regard this tumour as being a sclerosing hæmangioma. The later chapters deal with the reticuloses and are much less full than those on the other kinds of cancer. There is no mention of malignant changes occurring in chronic leg ulcers or of the rare sweat gland and sebaceous gland cancers. There is a magnificent international bibliography occupying thirty-seven pages, the only major omission being Dupont's important work on Kaposi's sarcoma. An appendix gives an account of the newest work which has appeared while the bulk of the book was being printed. The index is satisfactory though it points the omission of well-known synonyms such as Marjolin's ulcer and erythematoid benign epithelioma. There are many beautifully reproduced photographs and the production of the book is of high standard. This book is warmly recommended, especially to dermatologists, radiotherapists, and surgeons.

Cancer of the Pharynx, Larynx and Œsophagus. By Ronald W. Raven, O.B.E.(Mil.), T.D., F.R.C.S. (Pp. xiv+292+10; illustrated. 67s. 6d.) London: Butterworth & Co. (Publishers) Ltd. 1958.

In this well-produced and generously illustrated treatise emphasis is on surgery, mainly extensive surgery for advanced disease. The preface states that treatment should be based on detailed knowledge of anatomy and special pathology. One would add especial skill in diagnosis of the site and extent of disease in regions difficult to examine. It is perhaps for this reason that much of this work is undertaken by laryngologists in close collaboration with radiotherapeutic departments. For some time much of the earlier cancer of the larynx, almost all that of the nasopharynx, and certain areas of the pharynx have tended to receive radiotherapy as a first line of treatment. The advanced cases

and radiotherapeutic failures have become surgical responsibilities, with increasing success due to developments in anæsthesia, the combating of shock and prevention of sepsis.

While much of this is mentioned in parenthesis such a treatise should give full weight to alternative methods or combinations of methods, the personal predilection of the author then being emphasized.

The statement that adequate trained staff and facilities for radiotherapy are not universal and that surgery still may have a major part to play is unexceptionable. Such situations, however, are becoming rarer with the advent of rapid and cheaper transport to regional centres.

The book is of value as a record of success by the author's chosen methods, but can hardly be regarded as a textbook on the subject.

Vascular Spiders and Related Lesions of the Skin. By William Bennett Bean, M.D. (Pp. xix+372; illustrated. 63s.) Springfield, Ill.: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1959.

Dr. Bean's spiders are as fascinating as those of Henri Fabre. Observation, measurement, style and wit make a physician's and dermatologist's elixir. Dr. Bean has zealously pursued and tracked down angiomatous malformations over many years and has spun them together into an attractive yarn. He deals extensively with spider nævi and palmar erythema, discusses hereditary hæmorrhagic telangiectasia and then refers more briefly to a host of conditions relevant to his theme. The book is well produced, is in clear type making for easy reading, and is well illustrated by many good photographs of clinical conditions and by interesting diagrams. The bibliography is extensive. A work of erudition that is right up to date and that will make the physician look with renewed interest at the patients he sees every day even though some of the topics dealt with are uncommon. A suitable gift for a medical colleague.

Health in Industry. By Donald Hunter, C.B.E., M.D., F.R.C.P. (Pp. 288; illustrated. 4s.) Harmondsworth, Middx.: Penguin Books Ltd. 1959.

In the introduction to this book Dr. Hunter draws attention to the need to emphasize more to medical students the importance of social and preventive medicine. Knowledge of the recognized occupational diseases, and an appreciation of the fact that factors at work may contribute to or cause common ailments such as the neuroses, asthma, bronchitis, dermatitis, muscle strain and fibrositis, will stimulate the medical student or practising doctor to probe carefully into the occupational history of his patients and to discover that in many cases steps can be taken to prevent or to lessen the effects of occupation upon health.

This contribution to the Pelican Medical Series provides a source of information about industrial diseases and accidents which will be of value not only to doctors and medical students but to all who are in any way responsible for maintaining safe working conditions. A glossary is included to assist readers who are unfamiliar with the technical terms used in chemistry, physics, biology and medicine, and there is a comprehensive list of references and suggestions for further reading.

Many occupational diseases and the occupations which give rise to them are described in detail, as are the measures adopted for prevention of these diseases. The sections on the history of industrial medicine and legislation affecting the worker will be of interest to everyone. Although in some places the author has achieved condensation of facts at the expense of clarity and ease of reading, he has succeeded in presenting specialized knowledge in a manner which will interest readers from many walks of life.

Mental Illness in London. By Vera Norris, M.B., Ph.D. Maudsley Monographs No. 6. (Pp. 317; illustrated. 35s.) London: Chapman & Hall, Ltd. 1959.

The late Dr. Norris here sets out her survey of the hospital care of the mentally ill in London, in so far as this can be described by numerical data. The amount of work involved was enormous, for it was based on a sample of nearly 10,000 patients, all those admitted during 1947–1949 to two Observation Units and three Mental Hospitals; they were followed up, as far as possible, until the end of 1951. A great deal of statistical information is given in many clearly designed tables and figures; those who want even more are referred to the Institute of Psychiatry.

Even if this survey "cannot be expected to do more than give broad outlines of mental hospital practice, providing a background against which more intimate studies can be viewed" its value should not be underestimated by psychiatrists. In Dr. Norris' discussion of her findings much of interest emerges: for example, the figures support the view that observation units fulfil a useful function in large cities; less than one patient in four admitted to hospital for schizophrenia or manic-depressive psychosis, and discharged within two years, has not been readmitted within about four years. Even more would have been learnt had there been more comparable studies in the past. As it is, this work will be invaluable when attempts are made, for example, to assess the effects of the past decade's changes in mental

hospital care. The final chapter discusses, amongst other things, psychiatric classification, the incidence of mental disorders in different cultures, the influence of sex, age and marital status on mental hospital admissions, and prognosis, about which the figures suggest that other authors have been unduly optimistic.

This is an important work and a worthy addition to the Maudsley Monographs.

Treatment of Lung Cavities and Endobronchial Tuberculosis. By Beryl E. Barsby, M.D., M.R.C.P. (Pp. vi+147; illustrated. 20s.) Edinburgh and London: E. & S. Livingstone, Ltd. 1959.

This monograph is an expanded version of a successful prize essay submitted by the author to the British Tuberculosis Association. It comprises a study of the endobronchial manifestations of tuberculosis as seen in Malaya, and is based on the findings in 1,126 bronchoscopies. These findings have been classified into six grades of severity and a good account is given of the symptoms, signs, radiological appearances and treatment of the condition. The author has allowed herself to go outside the main theme and to discuss such immediate and remote consequences of tuberculous bronchitis as cavity formation, cavity persistence and tuberculous empyema. Her method of treating cavities in the type of disease with which she is confronted is most sensible.

Although acute progressive phthisis of the kind which forms the basis of her material is fortunately rare in this country, this monograph contains information which will be helpful to workers in many parts of the world. The author is to be congratulated on achieving such results under difficult conditions and on embodying them in a volume the text of which is lucid and the illustrations for the most part excellent.

A System of Orthopædics and Fractures. By A. Graham Apley, M.B., B.S., F.R.C.S. (Pp. vii+357+19. 47s. 6d.) London: Butterworth & Co. (Publishers) Ltd. 1959.

This book on orthopædic and fracture surgery provides most concentrated reading and very successfully includes a wealth of information in a relatively small space. The emphasis is placed on clinical diagnosis, with the practical application of "look, feel, move and X-ray" forming the background—the basis of which is clearly the teaching of Professor George Perkins and the St. Thomas's school. The three main sections in the book—General Consideration, Regional Orthopædic Surgery and Trauma—must naturally interdigitate, but the author has very cleverly avoided repetition by cross-reference. With suggestions for further reading at the end of each chapter an excellent bibliography is provided, so the more advanced student can use this book to summarize his knowledge and delve more deeply into the literature when and where he wishes with the greatest of ease.

By dispensing with pictures and diagrams the size and cost of this volume have been kept down; though this may tend to make dry reading in this type of book it is probably preferable.

Details of operative treatment are not included, but enough is stated to cover the salient features, so that the undergraduate reader is not subjected to material which he cannot fully comprehend, and postgraduates can find enough information to stimulate wider reading.

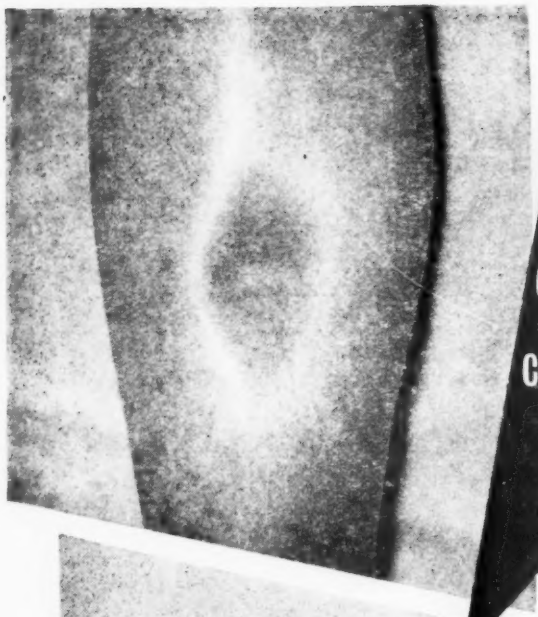
Throughout the text there is a tendency to be dogmatic, probably for the sake of brevity, at the cost of some accuracy, but this does not detract from the value of a book which will provide a wealth of classified information for the examinee.

Hearing. A Handbook for Laymen. By Norton Canfield, M.D. (Pp. 214.) New York: Doubleday & Company Incorporated. 1959.

This intensely interesting book concerning the problems of the hard of hearing is written for the layman and in most respects the advice given makes the task of the otologist easier. Dr. Canfield exposes the fact that it is impossible for the otologist to tell the patient what percentage of his hearing he has lost. In exchange he offers the patient a simple classification of his hearing loss, and this makes sense both to the patient and the otologist.

The reviewer's criticisms are as follows: Dr. Canfield appears to offer the patient more than otology or audiology can provide at present and many patients will be disappointed in the performance of their hearing aids after reading this book. It is suggested that there is a large psychogenic element in the ætiology of Ménière's disease and of otosclerosis; it is agreed that one cannot divorce psychology from any human situation, whether it be otosclerosis or a road accident, but we refer road accidents to the road engineer rather than the psychologist, and similarly we refer otosclerosis to the surgeon or the hearing-aid expert rather than the psychiatrist.

This book will undoubtedly help the hard-of-hearing public in the English-speaking world and in doing so will confer benefit on an enormous number of people. Dr. Canfield is to be congratulated in putting aside his vast knowledge of the subject so that he can see the problem from a layman's point of view and then in using this knowledge to indicate the best way of solving these problems.



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